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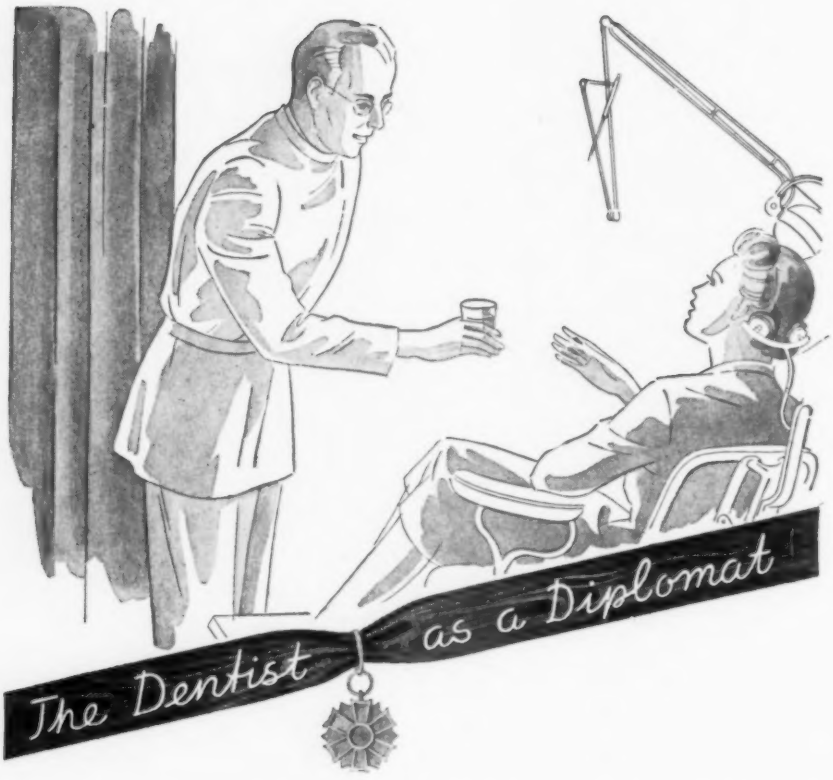

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Original Articles

EXPERIENCES IN EXODONTIA AND ORAL SURGERY AT AN ARMY POST

WALTER C. GURALNICK, 1ST LT., D.C., A. U. S.

EXODONTIA and oral surgery as practiced in the Army are little different from civilian practice. Primarily, the same type of work must be done and the same problems faced. There are slight differences: first, the need for greater speed in completing cases; and second, the advantage of being able to follow them more closely. Because of the great number of cases and the fact that end results can be seen, it is possible to arrive at some idea of methods which are expedient. Moreover, in dealing with numbers of patients, one integrates many small practical techniques which are time-saving and beneficial. It is the purpose of this paper to review some methods of everyday practice which are occasionally overlooked but, when used, are helpful. We will not attempt to discuss complicated operations, but rather incidental procedures which become very important in hastening the preparation of soldiers for combat duty.

Before treating patients, an efficient surgical setup is necessary. In any type of surgery, it is of paramount importance to maintain as great a degree of cleanliness and asepsis as possible. In our particular installation, we favor the use of a "wet" table (Fig. 1). After sterilization, all instruments are placed in trays of "Instrument Keep," a preparation essentially of phenol, glycerin, and alcohol. A pair of sterile tongs is kept handy for the removal of any desired instrument or other sterile equipment. Sponges are cut up and prepared by enlisted personnel and then sent, along with linens, to the operating room to be autoclaved. These are ordinary and standard procedures, but important ones in the chain of observing all necessary precautions in order to avoid postoperative infection.

ASEPTIC TECHNIQUE

For ordinary extractions, a routine aseptic technique is obviously unnecessary. In extensive surgery, however, such as alveolectomies or cystectomies, we favor an aseptic technique. The argument that the mouth is a contaminated field does not contradict the importance of an attempt at asepsis. Granted that even preoperative mouthwashes do not accomplish sterilization of the oral cavity, it is still important to prevent any further contamination by the operator. Sterile drapes and instruments, as well as scrubbing by the surgeon, obviate contamination. In Fig. 2 a patient is draped in the manner which we employ. The head wrap is a sterile towel fastened with a towel clip. The larger drape is one which was fashioned from ordinary muslin in the shape of a bib, with two strings attached for tying. A second sterile towel is placed over the bracket

From the Station Hospital, Morris Field, Charlotte, N. C.

table. The sterile technique, in selected cases, is no more time-consuming than ordinary methods, and it does minimize the possibility of introducing infection into incised and traumatized tissues.

ROUTINE EXTRACTIONS

Ordinary extractions are the most important part of our work. The advantage of doing them in an Army post is the ability to follow the patient's postoperative course. Being able to observe results closely presents the opportunity for evaluation of methods. One fact has impressed us, and that is the advantage of suturing after simple extractions, particularly of posterior teeth, and usually following the removal of more than one tooth. However, even after a single large molar is extracted, if there is any freeness to the lingual, or palatal, and buccal tissues, a single mattress suture across the socket will accelerate healing and add to the comfort of the patient. Where two or three teeth have been extracted, a few sutures, placed without difficulty and with little loss of time, are very important. Usually if two teeth have been

Fig. 1.

removed, one suture drawing the lingual and buccal interdental papillae together, will serve to give some approximation to an otherwise large and gaping wound. As we mentioned in the beginning, time of healing is important to us. The sooner an extraction wound heals and the less discomfort that is caused, the sooner a soldier can proceed with his normal duties, or prosthesis can be made for him. For this reason, we urge a greater consideration of suturing.

Carrying the thought a step further, we feel that in performing an alveolectomy, an operator can hardly err on the side of oversuturing.

ALVEOPLASTY

The approximation of the mucous membrane of the ridges, following an alveolectomy, is best done with many closely placed sutures. Well-approximated tissues heal in a minimum of time and are ready to receive prosthetic restorations sooner than those which are loosely sutured. The patient, moreover, has less postoperative discomfort, and if the sutures are removed in three or four days, the result is a clean mouth. We were first impressed by the importance of many sutures, following alveolectomies, when the practice was urged by a very competent prosthodontist. He emphasized the fact that in his experience such treatment produced ridges better suited to dentures. The opinion was further corroborated by observing methods of plastic surgery. In placing full thickness

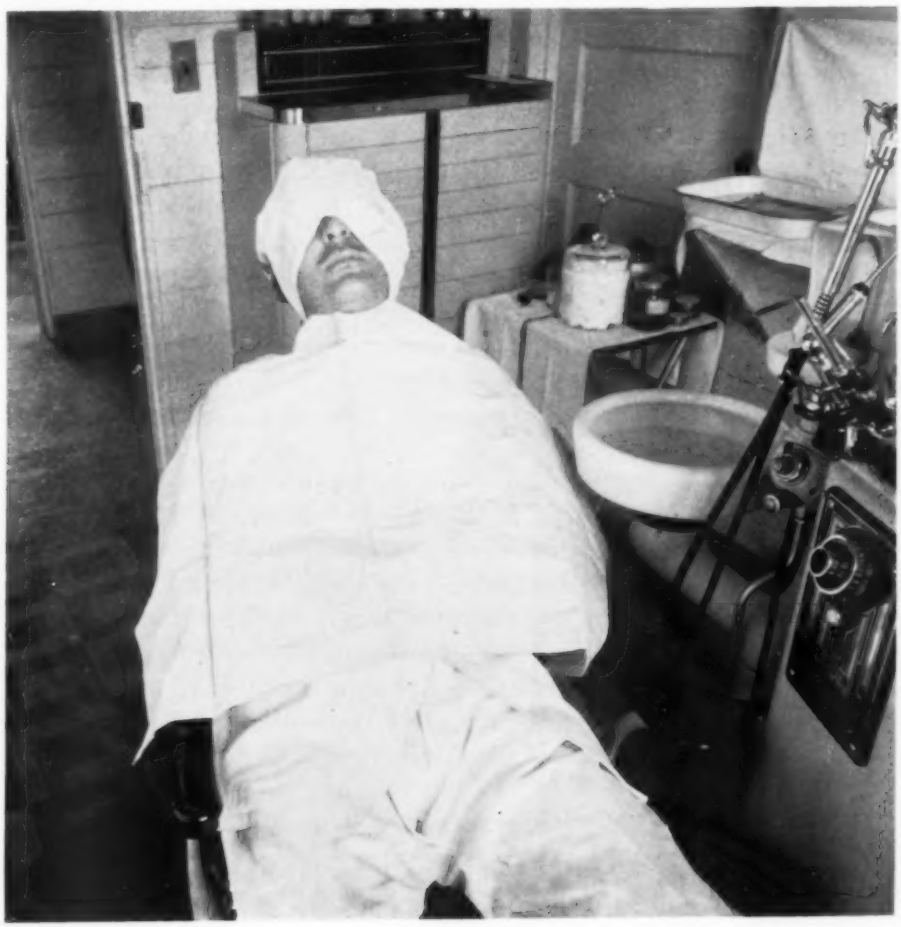

Fig. 2.

grafts, or approximating lacerations, innumerable sutures are taken. By the device a more cosmetic result is obtained in shorter time. The practice is as applicable to suturing after an alveolectomy as it is to the closing of facial wounds.

The analogy brings to mind the term "alveoplasty," which was coined some time ago. Alveolectomy was claimed to be a misnomer because the procedure is actually a plastic repair and not a removal of the alveolar process. The statement is quite true. It matters not what we call the operation, but it is important to bear in mind the nature of it, which is plastic. In fact, we improve results in mouth surgery by adopting as many as possible of the principles of plastic work. One principle, the use of many sutures, has already been mentioned.

Another helpful procedure is the removal of a triangular section of tissue from the mucous membrane on the distal of the tuberosity, if an alveolectomy is carried back that far. The removal of the segment allows buccal and palatal tissues to be well approximated. It also prevents a flabby tuberosity from resulting in the edentulous area. The operation is very much analogous to the method of sliding flaps to close gaps in other parts of the body surface. The procedure, as applied in the mouth, was very aptly described by one man as "cutting out a piece of pie." It is usually all that is necessary to get good approximation. In certain cases, however, this is not sufficient in itself. In some mouths an extremely thick palatal mucosa is encountered. In such cases it is almost impossible to slide the tissue, even after it is undermined. Lt. Col. Hemberger, at the Walter Reed Hospital, pointed out a simple remedy. The palatal tissue is split, not deeply, but enough that the newly thinned portion can be stretched. The inner layer can be removed or it may be left, if desired, as long as the purpose of obtaining a movable tissue is accomplished.

There is another simple procedure which we have found expedient and helpful, a method for removal of a buccal protuberance in the tuberosity region, which often forms an undercut almost impossible to cover with a denture. A simple and efficient method for removal of this troublesome prominence was once demonstrated to us. A sharp, broad chisel (about $\frac{5}{8}$" in width) is placed at the anterior side of the prominence. The vertical tilt of the blade is such that it is in harmony with the normal ridge direction. The chisel is now malleted, its sharp blade cutting through the mucous membrane and, at the same time, raising a flap in which there is the excessive bone of the prominence. This bone is now easily detached from the flap, and the mucous membrane is allowed to fall back into place, where it remains in place equally as well with or without suturing. It is obvious that the operation can be done in a few moments. The simple procedure, moreover, is followed by extremely rapid healing of the little-traumatized tissues, and prosthetic work can be started almost immediately.

POSTOPERATIVE CARE

In spite of the refinements of technique, if tissues are not handled with care and respect, postoperative results are disappointing. One of the usual causes of undue trauma is over-retraction. Exposure of the operative field is certainly desirable, but the surgeon must sometimes forego ideal conditions for himself in the interest of respect for the patient's tissues. The patient's comfort is also affected by the postoperative orders he is given.

Fig. 3 is an illustration of the instructions given to our patients following any extensive procedures. In addition, the soldier may be given an assignment

to quarters for twenty-four or forty-eight hours, or be hospitalized if it is deemed necessary after a lengthy and tiring operation. The combination of rest and relief from pain goes far toward speedy and uneventful recovery. As to the immediate use of ice packs, we are aware of the literature which disclaims its value. Clinically, however, immediate application of ice, as prescribed, seems to minimize swelling. The same instruction sheet is given to all patients following even a single extraction. For the latter cases, items 3 and 4 are struck out. Since no discomfort or swelling is anticipated, it is essential to impress upon the soldier only the measures necessary for the control of bleeding.

INSTRUCTIONS

1. Bite on gauze for 20 minutes, then throw it away.
2. Do not rinse today.
3. Apply ice pack—keep in place for 15 minutes, remove for 15 minutes. Continue for several hours. (Ice can be procured from your mess.)
4. Take one capsule* every 4 hours if you have pain.
5. TOMORROW: Rinse every two hours with a glassful of hot water with ½ teaspoonful of salt in it.
6. Return to clinic if you have undue pain, bleeding, or swelling.

*Composition of each capsule mentioned above is:

Aspirin	3.3 gr.
Phenacetin	2 gr.
Caffeine citrate	½ gr.
Codeine sulfate	¼ gr.

Fig. 3.

FLAPS AND DRESSINGS FOR CYSTS

One other technique which we favor is the use of the semilunar flap for eradication of cystic or granulomatous areas in the anterior part of the mouth. The depression resulting from loss of anterior buccal plate is a familiar sight. This can be avoided if the semilunar flap, almost always used for apicoectomies, is substituted for the usual flap technique in the vestibule of the mouth. The designated tooth is extracted, and the flap is then turned. Ordinarily, the buccal bone over the infected area is necrotic and easily punched through. The window can be opened enough to give sufficient access for complete enucleation of the cavity, and at the same time a bridge of buccal plate is left intact between the gingival margin and the cavity. The preservation of even a small amount of bone here is enough to maintain the normal contour of the alveolus.

A word might be added about the removal of cysts. Whether or not to close a flap tightly following the removal of cysts is usually governed by the presence or absence of suppuration in the wound. Following the removal of an acutely infected cyst, a drain is left in the cavity for some time. The "clean" cyst cavity is closed completely. We feel that the presence or absence of pus is not the only criterion by which to be governed. The more important consideration is the size of the cyst. A small infected cyst, about the size of a large pea, can be completely removed, and the mucous membrane may be tightly

closed with impunity. However, if complete closure is effected after the removal of even a nonpurulent but large cyst, secondary infection is apt to result in the area. We, therefore, favor packing large cyst cavities. Instead of suturing the incision tightly, we leave enough of an opening that a greased wick can be introduced and lightly packed into the cavity. The dressing causes no discomfort to the patient. It requires only infrequent changing, due to the fact that as the wound granulates in at its base, the greased wick is expelled. The initial dressing can be left in place for five or six days, and when the patient returns for the removal of sutures, a smaller wick may be substituted for the original one. After that, at five- or six-day intervals, the dressing may be shortened as it becomes extruded from the wound, or fresh and progressively smaller dressings can be substituted at visits about a week apart. In approximately three weeks, when a healthy bed of granulation tissue has formed over the surface of the bony cavity, dressings can be dispensed with, and the healing process will continue successfully. A cystectomy is one operation in which a speedy end result is not always of prime importance.

In the repair process, we mentioned the use of a greased dressing. Instead of ordinary vaseline gauze, we use a dressing material which seems efficacious. Its virtues are its vaseline content and, at the same time, an obtundant action. At the oral surgery clinic at the Boston City Hospital, a preparation called "magic" has been used for many years in the treatment of painful or "dry" sockets. The connotation of the name is bad, but the drug is actually extremely effective. It consists of iodoform, 21 grams, guaiacol and chloroform, 8 ounces each, to make one pint. We incorporate the solution into petrolatum. Strips of gauze are then autoclaved with the medicated vaseline, and the gauze becomes impregnated with petrolatum and "magic." We use it for dressings following the removal of cysts and also as a dressing material in painful sockets. With apologies for its name, we find the medicine very effective in alleviating pain.

SUMMARY

We have attempted to review some methods of treatment used in our everyday practice of oral surgery at an Army installation. The techniques mentioned are as applicable to civilian practice as to military. Essentially, scrupulous cleanliness, careful technique and attention to postoperative care of the patient are the aims of the Army or civilian surgeon. Routine attention to detail is the best prophylaxis for postoperative infection and discomfort. We should appreciate the good fortune of working in so resistant a field as the mouth and take care not to abuse its remarkable tolerance. Because of the unique healing powers of the mouth, if we but practice slight refinements in oral surgery, results can be very gratifying.

A PRACTICAL OUTLINE FOR THE TREATMENT OF BURNS

DONALD W. MACCOLLUM, M.D.,* BOSTON, MASS.

FOR several years before the present war, the number of people in this country who died each year from burns ranged in the vicinity of 6,000. In addition, 1,500 died each year as a result of conflagration, and untold thousands lived but were permanently disfigured or deformed. Since burns are not acquired or inherited like disease, but are almost always purely accidental, more and more rigid safety measures have been instituted both in the home and in industry in an attempt to reduce these figures. Obviously, if fewer people were burned, there would be fewer deaths, but prevention is only one of the two significant factors concerned in this high death rate. The other, equally important, factor is a proper appreciation of how to handle the burned patient from a medical standpoint. To lessen the incidence of confused, unintelligent, and inadequate care by the layman, nurse, and physician alike would effect an appreciable reduction in the number of fatalities and deformities caused by burns.

During peacetime, 83 per cent of all burns occur in the home, 10 per cent in industry, and 7 per cent in public accidents. It is obvious from these figures that there is a great need for both an educational program and the elimination of physical hazards in every home. This is to be kept in mind now and after the war is over. However, in wartime, with the possibility that explosive and incendiary bombs will drop on civilian rather than on military lines of defense, the number of burn casualties is in danger of being greatly increased. This has been true in every country that has experienced any number of air raids. For this reason, every person should have some knowledge of what to do and, perhaps more vital, what *not* to do for a person who has been burned.

The Red Cross and the Office of Civilian Defense have accepted and are discharging a tremendous responsibility in training a large number of laymen in the rudiments of first aid. Every physician in the country has also been preparing and refreshing his mind, so that he will be equipped to take care of war casualties.

In line with this general preparation, the following condensed outline for the treatment of burns has been drawn up, based on experience both personal and collective, to aid in the rapid assimilation of the present knowledge on this subject.

I. THERMAL BURNS (HOT LIQUIDS, STEAM, FLAME AND HOT METALS)

1. First-Aid Instructions for Laymen.
   a. Treat all burned patients for shock.

Presented in part at the annual meeting of the Massachusetts Medical Society, Boston, May 27, 1942.

From the Department of Surgery, Harvard Medical School, and the Surgical Service of the Children's Hospital and of the Peter Bent Brigham Hospital.

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*Associate in surgery, Harvard Medical School; associate visiting surgeon, Children's Hospital; associate in plastic surgery, Peter Bent Brigham Hospital.

b. If a physician is not present but is available:
(1) Cover small burns with a clean cloth or sterile bandage, and take the patient to the first-aid post.
(2) For extensive burns, cover the patient with a clean sheet and blankets; bring the doctor to the patient, if possible—otherwise send the victim to a hospital immediately.
(3) Do *not* put tannic acid ointment on any burn.

c. If a physician is neither present nor available and it seems likely that one or two hours may elapse before the patient can be seen by a physician:
(1) For small burns, clean the area by gently washing with a mild soap under running water—if running water is not available, do not clean, but cover the burn with a paste of sodium bicarbonate and water; wrap the burned area securely in clean or sterile cloths, and send the patient to a physician.
(2) For extensive burns, remove the clothing but do not try to clean the area; cover the burned parts with clean cloths (napkins, pillow cases, or sheets) soaked in a warm solution of sodium bicarbonate—cover with dry cloths; keep the patient warm and get him to a hospital as soon as possible; give strong coffee or tea by mouth.

d. Important points to be remembered:
(1) Do *not* apply tannic acid jelly or strong tea to a burn until after the burn has been properly cleaned by a doctor.
(2) Do *not* apply grease in any form.
(3) Do *not* move the patient about in an attempt to put on a neat bandage; disturb him as little as possible.

2. First-Aid Instruction for Physicians.

a. Check all patients with minor or major burns for shock; treat this even before the burned area is examined.

b. Give morphine liberally: administer a dose double that which would normally be used for the patient if he were to have a preoperative dose for an elective operation.

c. If shock is severe, administer oxygen by mask or tube and give caffeine intramuscularly; if plasma is available, give 5 c.c. per pound of estimated body weight intravenously; if plasma is not available, give physiologic saline solution—10 c.c. per pound of body weight—while plasma is being obtained.

d. When shock has been effectively combated, carefully remove the bandages and clothes; examine the burn and try to estimate extent and depth of the burn.

e. If the burn is minor:
(1) Cleanse thoroughly by washing gently for about seven minutes with sponges soaked in 50 per cent green soap (solution, not tincture) and 50 per cent hydrogen peroxide.
(2) Break all blisters, and remove all dead skin and debris (use sterile instruments).
(3) Flush with copious amounts of physiologic saline solution, or distilled water, and dab dry.
(4) Dust the area with sulfathiazole, sulfadiazine, or sulfanilamide powder.
(5) For burns of the hands, feet, face, and genitalia, apply a sterile ointment and cover as well as possible with sterile gauze and bandage.
(6) For burns elsewhere on the body tannic acid jelly or triple-dye jelly may be used; cover with sterile gauze, apply an Ace bandage, and splint the part involved.

f. If the burn is major:

(1) Remove the clothes sticking to the burn and gross dirt or contamination, but do *not* clean *extensively*.

(2) Dust the area liberally with sulfathiazole, sulfadiazine, or sulfanilamide powder (if a 5 per cent sulfadiazine, water-soluble jelly or emulsion is available, this may be used instead of the powder).

(3) If the powders are used, cover the area with a liberal amount of water-soluble glycerin-tragacanth jelly (if this jelly is not available, any sterile ointment or triple-dye jelly can be used; however, the latter are harder to remove when the burn receives its final débridement).

(4) Apply heavy (5 to 7 cm. thick) sterile gauze pads and hold them securely in place under mild pressure with an elastic bandage.

(5) Evacuate the patient to a hospital as quickly as possible.

g. Important points to be emphasized:

(1) Do *not* fail to appreciate the fact that shock must be treated *first* and that examination of the burn should wait until the patient is fairly well recovered from the shock.

(2) Do *not* apply tannic acid in any form until *after* the burn has been *thoroughly* cleansed; never apply it to the hands, face, feet, or genitalia.

(3) Do *not* temporize with morphine—give it either in a large enough dose to do some good or not at all.

(4) Do *not* allow the sulfonamide powders to get into the eyes or up the nose (they irritate as foreign bodies).

(5) Do *not* fail to apply a *thick* dressing under mild pressure.

3. Hospital Treatment of Fresh Burns.

a. For minor burns treat the patient as outlined above (Section I, 2*e*, 1-6).

b. For major burns:

(1) Check on the degree of shock still present; administer more morphine, heat and so forth, as required.

(2) If time permits and facilities are available, obtain a red-cell count, hemoglobin determination, hematocrit reading and a serum protein (one or more of these are helpful in determining the degree of hemoconcentration).

(3) Give an initial intravenous infusion of plasma of 3 to 5 c.c. per pound of estimated body weight; have more plasma available if it is needed; whole-blood transfusion is not indicated unless there has been appreciable blood loss from some other source; if whole blood is available but no plasma, make an effort to separate the plasma from the red cells by centrifugalization or otherwise.

(4) After shock is well controlled, the patient may need to be anesthetized for proper cleaning; use rectal or intravenous anesthetics for those who may have inhaled hot air or gases.

(5) Wash burned area thoroughly for seven minutes with a mixture of solution of green soap and hydrogen peroxide (equal parts).

(6) Remove all dead skin and debris; use sterile precautions throughout débridement.

(7) Flush thoroughly with physiologic saline solution and, if desired, some mild antiseptic, such as a 1:1,000 solution of Zephiran or Merthiolate, or S.T. 37.

(8) For burns of the hands, feet, face, and genitalia, dust the area with one of the sulfonamide powders and cover with a sterile ointment (*tulle gras*) and a heavy layer of gauze sponges held in place by an elastic bandage; splint the hands and feet.

(9) For burns elsewhere on the body, the treatment outlined in Section I, 3*b*, 8, *may* be used, provided adequate facilities are at hand to combat fluid loss by repeated plasma transfusions.

(10) For burns elsewhere on the body, the escharotics are *usually* employed, principally 5 per cent tannic acid followed by 10 per cent silver nitrate, the triple dyes or a sulfadiazine spray; whichever method is used, specialized aftercare is required: a clean or sterile electrically heated bed, frequent evaluation of the blood chemical findings, adequate fluid intake enterally and parenterally, and constant nursing and medical appreciation of the problems for each case.

4. Hospital Treatment of Old Burns.

a. If a sulfonamide powder has been used in the first-aid treatment, a débridement as outlined in Section I, 3*b*, 4-7, may be carried out as late as thirty hours after the burn was received.

b. If a sulfonamide has not been used, a complete operative débridement is contraindicated if the burn is eight or more hours old. In this event, the following procedure is advised:

(1) Give shock treatment as outlined in Section I, 3*b*, 1-3.

(2) Immerse the patient in a tub of warm tap water for one or two hours, with constant inflow and outflow of water.

(3) Remove the patient from tub, dab dry and spray with one of the sulfonamide powders.

(4) Either cover the burns with an ointment (preferably *tulle gras*) or use the triple-dye jelly or sulfadiazine spray; do *not* use tannic acid in any form.

(5) As an alternative to the procedure outlined immediately above, one may use dressings wet with physiologic saline solution, a Bunyan envelope or *tulle gras* with Eusol (12.5 Gm. chlorinated lime, 12.5 Gm. boric acid, and 1,000 c.c. distilled water—let the mixture stand twenty-four hours, then filter and keep in a tightly corked bottle); if the burn is coated heavily with fibrin and pus, one of the latter is preferable *first*, to be followed later by a sulfonamide powder.

c. In second-degree burns, healing takes place without grafting over a period of ten days to two weeks.

d. In either superficial or deep third-degree burns (so-called "third-degree," "fourth-degree" and "fifth-degree," according to one system of classification), grafting may be necessary, depending on the extent and site involved.

e. If grafting is necessary, certain precautions must be observed before proceeding:

(1) The patient must be free from infection, in good health, and with a normal red cell count.

(2) An estimate of the amount of tissue to be replaced must be made, and a program outlined for its transfer.

(3) The granulations must be flat, firm, and cherry pink.

(4) The granulations should be removed down to the fibrous-tissue base from which they spring, and depending on the site involved, this fibrous base may also be removed.

(5) For defects involving the loss of skin only, a thin or thick razor graft (Thiersch or split Thiersch) is preferable.

(6) For small areas involving the skin of the face or hands, a full thickness graft (Wolfe or Wolfe-Krause) may be used interchangeably with the thick razor graft.

(7) For areas involving the loss of both the skin and subcutaneous tissue, a single-hinged or double-hinged pedicle flap (pedicle, gauntlet, tube, or rope) must be used.

(8) Bone, cartilage, and fascia may also be replaced, but this is usually done six to eight months after the skin defects have healed.

(9) Do *not* use island grafts (pinch, Reverdin, small deep and Davis grafts) unless nothing else is possible; there is seldom occasion for their use.

II. CHEMICAL BURNS

(Whoever treats chemical burns should take adequate precautions to protect himself)

1. Mustard Gas.

a. First aid for exposure to mustard-gas vapor (one to eight hours).

(1) Remove and place all clothes in decontamination bucket.

(2) Give a thorough soap-and-water bath, including shaving or clipping short of all hair and rewashing these areas.

(3) Apply a bleaching-powder wash, followed by a second soap-and-water cleansing.

(4) Keep the patient under observation for twelve hours; if there is no rash or blisters at the end of that time, the patient may be discharged.

b. Late treatment for exposure to mustard-gas vapor (eight hours and over):

(1) Treat the patient for shock; rash and blisters have appeared over warmed areas of the body (axilla, groin and so forth), with severe itching and beginning shock.

(2) Do *not* sedate heavily with morphine (pulmonary complications due to inhalation of the gas may be increased by a lowered respiratory rate).

(3) Anesthetize by the rectal or intravenous route, if necessary.

(4) Clean the burn thoroughly, as outlined in Section I, 3*b*, 5-7.

(5) Apply *tulle gras* over all raw surfaces and follow with a heavy dressing wet with equal parts of Eusol and mineral oil.

(6) Apply a heavy dressing, held in place with gauze and an Ace bandage under mild pressure.

(7) Follow the routine outlined for the aftercare of second-degree or third-degree thermal burns (Section I, 4*c*, 1-9).

(8) For very late infected cases seen two or three days after exposure, follow the routine outlined in Section I, 4*b*, 1-3, 5.

c. First aid for contact with mustard-gas liquid.

(1) Within five minutes, *blot* off excess mustard gas by dabbing with dry pads.

(2) Dab the area gently and repeatedly with pads moistened in kerosene, gasoline, or alcohol; wring the pads out so that excess liquid will not drip on the patient and spread the burn.*

(3) Thoroughly wash the entire area with a mixture containing equal parts of green soap and peroxide.

(4) Wash the area again with alcohol.

(5) Dry by blotting, and apply a dry dressing that is to be removed and the area examined every two hours for the next twelve hours; if the area looks normal at the end of twelve hours, the patient may be discharged.

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*Because the mustard gas oil may be spread over a larger surface in using a solvent, it is now thought that immediate use of sodium hypochlorite solutions is better than kerosene, gasoline, or alcohol. Sodium hypochlorites should *not* be used after blisters form.

d. Late treatment for contact with mustard-gas liquid.

(1) If blisters have formed, dab with pads of kerosene gently to get rid of excess liquid; do *not* use a bleaching agent.

(2) There is no need to sedate heavily or anesthetize (the area is usually painless after liquid has penetrated through the skin).

(3) Carry out full débridement as outlined in Section I, 3*b*, 5-7, and follow with a *tulle gras* and Eusol dressing.

(4) In very late cases when the blisters have broken and have become infected, carry out the routine outlined in Section I, 4*b*, 2, 3, 5.

(5) In the late cases, as soon as the fibrinous infected coat has been removed from the raw surfaces by the Eusol, the sulfonamides may be used.

(6) Late care, including grafting, is the same as that for thermal burns.

2. Lewisite.

a. First aid for exposure to lewisite vapor.

(1) Remove clothing at once.

(2) Wash entire body with hydrogen peroxide; follow this with a complete and thorough bath with soap and water and an alcohol sponging.

(3) Keep the patient under observation for 12 hours; if no blisters appear, he may be discharged.

b. Late treatment for exposure to lewisite vapor.

(1) Sedate and give a rectal or intravenous anesthetic if burns are extensive.

(2) Wash around and over each blister with soap and hydrogen peroxide; be very gentle.

(3) Break the blister, being very careful to catch all the blister fluid on a sponge (throw sponge in decontamination bucket); immediately apply a pad wet with hydrogen peroxide to the raw area; renew this pad frequently.

(4) After all blisters have been broken and covered with a pad of hydrogen peroxide, carry out full operative débridement as outlined in Section I, 3*b*, 5-7.

(5) Cover the burn with a *tulle gras* and Eusol dressing, thick gauze and an Ace bandage.

(6) If the burn remains clean, reapply the dressing described in Section II, 2*b*, 5, each day.

(7) If the burn becomes infected, the treatment is the same as that outlined in Section I, 4*b*, 2, 3, 5.

c. First aid for exposure to lewisite liquid.

(1) Within one minute after exposure remove the clothes and start treatment.

(2) Swab area with hydrogen peroxide repeatedly for thirty minutes.

(3) Wash with soap and water for seven minutes, and follow with an alcohol pad, which is left in place for five minutes.

(4) Pat dry, and apply a dry dressing; if no blisters have formed at the end of twelve hours, the patient may be discharged; if blisters have formed, carry out the routine outlined in Section II, 2*b*, 2-4, except that anesthesia is usually not necessary.

d. Late treatment for exposure to lewisite liquid.

(1) If an unbroken blister is found, carry out the routine outlined in Section II, 2*b*, 1-7.

(2) If the blister has broken and the patient shows signs of arsenical poisoning excise entire area under anesthesia at once, and follow by grafting as outlined in Section I, 4*e*, 5-8.

(3) If the area is too extensive for excision and is grossly infected, the prognosis is poor; however, treat the patient as outlined in Section I, 4*b*, 2, 3, 5 and attempt to control the arsenical poisoning by intravenous sodium thiosulfate.

3. Phosphorus.
   a. If there is no phosphorus that is still burning in or on the skin, treat the burn as a thermal one (Section I).
   b. If bits of phosphorus that are still smouldering lie on or in the skin:
      (1) Wet the area and keep it wet with anything, but preferably warm water (this melts and stops further burning of the particles; if the area becomes dry, the particles will begin to burn again).
      (2) If a 5 to 15 per cent solution of copper sulfate is available, apply this at once (burning is stopped temporarily).
      (3) No matter which of the above methods is used, squeeze or pick out the phosphorus particles with forceps and then treat as any thermal burn.
4. Acids and Alkalies.
   a. Remove the patient's clothing at once, taking care that the chemical agent does not burn others.
   b. Flush area under cold running water (the heat of dilution of strong chemicals is carried away by the continuous washing); if running water is not available, douse the patient in a tank, tub, or lake; if the burn is due to lime, brush off the excess before wetting.
   c. Do not attempt to neutralize the burning agent (the heat of reaction may cause a deeper burn, or the neutralizing agent itself may burn).
   d. After removal of the burning agent, treat as a thermal burn (Section I).

III. ELECTRICAL BURNS

1. General Effects.
   a. Do *not* touch a patient who is still in contact with the electric current, unless proper insulation is possible.
   b. Never regard a recently shocked person as dead, even if the heartbeat cannot be heard.
   c. Continue artificial respiration, oxygen and cardiac stimulants until rigor mortis sets in (several patients have recovered after having been apparently dead for hours).
2. Local Effects.
   a. Except for a very few cases almost all electrical burns are of third degree owing to the intense heat and the coagulation of blood in the vessels supplying the area involved.
   b. Do *not* be misled by the apparently innocuous appearance of the fresh electrical burn; institute treatment at once:
      (1) Sedation or anesthesia is rarely necessary.
      (2) Clean the area thoroughly with a mixture of green soap and hydrogen peroxide and flush with saline.
      (3) Apply a sterile ointment, gauze, and an elastic bandage; splint the part involved.
      (4) Do *not* apply any substance that will form an eschar.
      (5) Apply daily dressings under strict aseptic precautions so that the amount of late necrosis and slough may be observed.

(6) Do *not* cut away sloughing tissue; allow the demarcation and detachment of dead tissue to take place without surgical aid (hasty operative removal may result in excision of tissue that will be viable and necessary for repair).

(7) Vessel walls are damaged, so that secondary hemorrhage is frequent; have a sterile tray with hemostats and a tourniquet always at the bedside.

(8) After the slough has disappeared, treat as in Section I, 4*e*, 5-8.

A general survey of the treatment of burns has been outlined, with particular emphasis on the treatment of shock, the need for gentleness in the handling of tissues, and the necessity for a complete cleansing of the burned area before medicaments are applied. Each burn presents an individual problem; consequently, it is impossible to outline specific details for every possible type of burn. The broad general principles have been stressed, in addition to specific instructions that, if followed within reason, will cause an appreciable reduction in the number of fatalities.

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THE ROLE OF THE SULFONAMIDES IN DENTISTRY

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WHEN the history of medical science is written, the present era will be marked as a great turning point. For this is the era of the sulfonamides. No chemical specific since the advent of salvarsan has promised so much. Certainly, no other chemical has faced as critical an appraisal. And, from the most recent reports, the sulfonamide group is living up to expectations.

Not unlike his medical confreres, the dentist applies the sulfa drugs systemically as well as locally, prophylactically as well as therapeutically. It has been shown that the sulfonamides are especially effective against the beta hemolytic streptococcus, *Streptococcus viridans,* and the *Staphylococcus aureus* organisms. These bacteria are commonly identified with dental sepsis. Thus, the drugs have come to play an important role in the therapy of the acute alveolar abscess, osteomyelitis of the jaws, cellulitis of the face or neck, or general sepsis of dental origin. Their use here is the same as for infections elsewhere in the body, and the same fundamental rules must be observed. It must always be remembered that the sulfa drugs are a weapon, powerful and effective it is true, but not self-sufficient. In the present armed conflict, the might of air power is important enough to make the difference between victory and defeat; but the air force must have the cooperation of infantry, artillery, mechanized units, units of supply, etc. So too, in the fight against infection, the fundamental medical and surgical procedures must not be slurred or omitted. Adequate drainage must be established where needed. The source of the infection should be eliminated as soon as possible. And proper supportive measures should be instituted immediately—bed rest, adequate diet, and thorough elimination of wastes and toxins.

Legally and technically, members of the dental profession have the right to prescribe and use the sulfonamides for conditions arising from dental and oral infections. However, no dentist or physician should avail himself of this privilege unless he is thoroughly acquainted with the nature of the drugs, their clinical pharmacology, and, especially in this group, their toxicity. Routine laboratory studies of the blood and urine should be performed during the course of chemotherapy. And regular medical attention should be available to check for clinical signs of toxicity to the drugs. It would be best, therefore, for the dentist to administer the sulfonamides in collaboration with a qualified physician. Hospitalization would be ideal, although in many cases impracticable.

In this respect, the dentist may be the first to note toxic manifestations of sulfonamide therapy, when these changes occur in the mouth. Their appearance is similar to that of a herpetic stomatitis, with the formation of small papules on the tongue, lips, cheeks, or palate, and occasionally involving the gingivae

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as well. The papules break down to form shallow ulcers, which tend to coalesce, forming larger designs. They disappear promptly upon discontinuance of the drug, but will reappear if that same drug is administered. The patient is usually not sensitive to other members of the sulfonamide group, and therapy may be maintained with a different drug (Figs. 1 and 2).

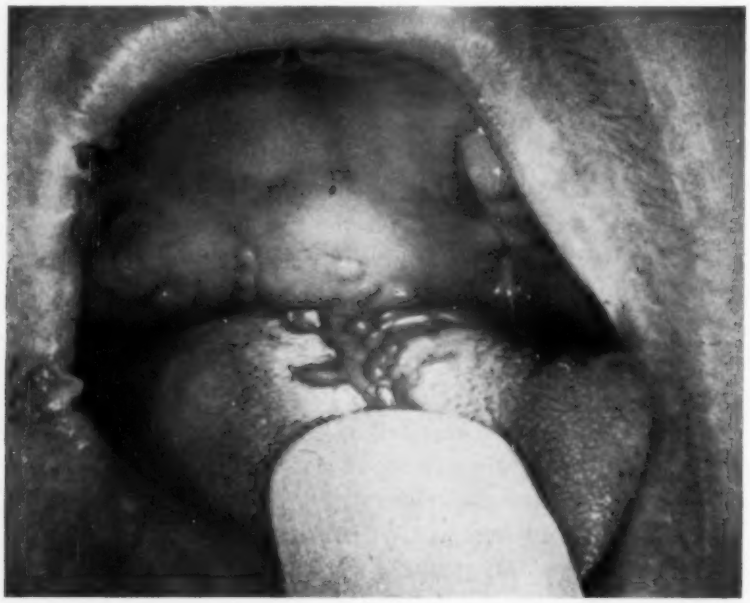

Fig. 1.—Oral manifestations of toxicity to sulfanilamide. Note shallow ulcerations on dorsum of tongue, palatal mucosa, and lips. The buccal mucosa and gingival tissues were also ulcerated and inflamed. All signs faded dramatically after discontinuance of chemotherapy.

A survey of the recent dental literature shows almost unanimous agreement as to the benefits of the systemic administration of the sulfonamides, and especially sulfathiazole, in the therapy of dental and maxillary infections.[1, 2] Occasionally, a toxic reaction has developed, and the drug was discontinued or changed to another in the sulfa group. It has also been reported that the sulfonamides have delayed localization and fluctuation by slowing the course of an infection. But these cases are rare. So much for systemic sulfonamide therapy. Its application to the dental field does not differ materially from its use for infections in other parts of the body.

The *prophylactic* value of establishing an effective preoperative blood level of the sulfonamides can be great. However, we are dealing with a potentially dangerous drug. The great majority of dental and oral operations are not followed by sepsis. We also have another, less dangerous, prophylactic weapon—the local application of the sulfonamides. For these reasons, routine preoperative medication has been rejected.

However, there is one group of cases in which the establishment of an effective preoperative blood level is important. It has always been the bugaboo of the oral surgeon that a patient with previous damage to the heart valves may develop subacute bacterial endocarditis subsequent to tooth extraction or almost any form of dental manipulation. It has been demonstrated repeatedly that the extraction of a tooth, or even the scaling of root surfaces, may set up a tran-

sient bacteriemia.[3-6] The organism recovered is almost always *Streptococcus viridans.* As it courses through the blood stream, the damaged heart valves offer an inviting site on which to vegetate and multiply.

Subacute bacterial endocarditis is usually fatal. Even the sulfonamides are rarely, if ever, of curative value. But as a prophylactic measure, they promise a great deal. The microorganisms pumped into the blood stream during manipulative procedures can be easily and quickly overcome before they can lodge deeply and become protected from the active circulation and the various body defenses.

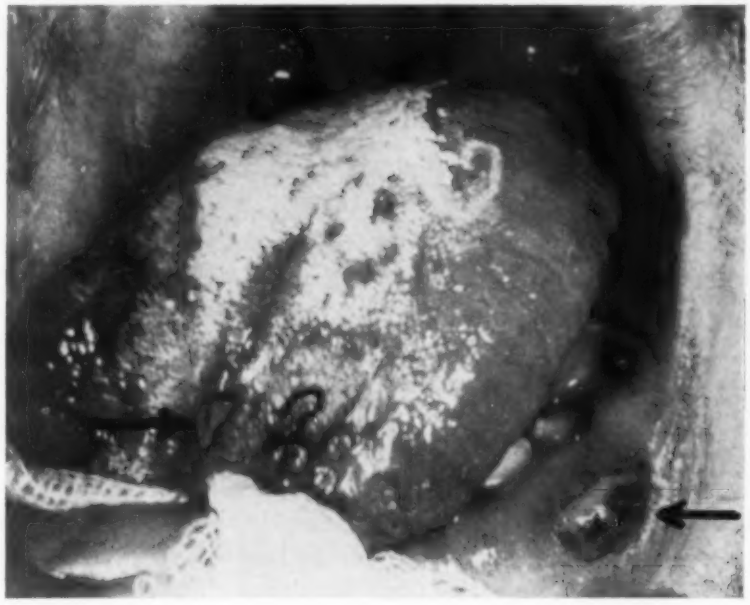

Fig. 2.—Oral manifestations of toxicity to sulfapyridine. The lip and tongue show ulcerative changes (arrows).

Thus, many institutions have adopted the following procedure. All patients requiring extractions who give a history or manifest clinical signs of damaged heart valves due to rheumatic fever, syphilis, or to a congenital defect are hospitalized. After proper workup, they receive enough sulfathiazole to establish an effective blood level of the drug. The operation is then performed, and chemotherapy maintained for twenty-four to thirty-six hours postoperatively, and then discontinued. Many of these cases have been followed for more than two years postoperatively, and there have been no signs of further cardiac damage attributable to the manipulative procedure.[4, 7, 8] From the indefinite nature of a clinical study of this type, it is impossible to draw any definite conclusions at present. However, the groups that have been pursuing these studies report very favorable results in lowering the incidence and virulence of bacteriemias following tooth extraction.

In studying the *local* uses of the sulfa drugs, we come to a much more controversial field. We shall not discuss the mass of claims and counterclaims, or the experimental and clinical reports which in several instances directly contradict one another. Out of the welter of confusion, certain facts and principles seem to stand out. These emphasize the fundamentals of tissue physiology and

surgical technique, and discredit the uncontrolled and indiscriminate use of the sulfonamides as a substitute for sound surgical procedures.

The sulfa drugs have been used locally to treat burns, ophthalmic conditions, and infected wounds in general. They are also used to prevent infection in surgical and traumatic wounds. Their effect is attributable to their low solubility and slow absorption and the resultant high concentration locally—up to 800 mg. per 100 c.c. The modern treatment of war wounds has been revolutionized. It has become accepted procedure first to perform early and complete débridement of the wound; second, to keep the part covered and absolutely at rest, preferably by a plaster cast. Supplementary local and general sulfonamide therapy has proved to be of definite value here, especially in the prevention and treatment of gas gangrene.[9]

In general surgery, the suspicion of contamination calls for the sprinkling of powdered sulfonamide into the wound before it is closed. It has been found that the use of a sulfanilamide or sulfathiazole paste, suspension, or solution for the irrigation of wounds has markedly decreased the incidence of secondary infection and has brought about rapid, uneventful healing. Of course, if an infection is due to an organism such as the tubercle bacillus, against which the sulfonamides have not been shown to have any specific effect, we cannot expect the use of the sulfa drugs to influence the course of that infection. We can hope that their presence will prevent the secondary invasion of already weakened tissues by other pathogens which may further complicate the picture.

The principles of treating wounds of the mouth are fundamentally the same as those applied to other parts. The most important difference is found in the fact that every extraction socket, every incision for whatever reason, is a contaminated wound. Every fractured jaw in the line of a tooth is compounded into the mouth. And, bacteriologically, the mouth is a filthy cavity. The wonder is that so few of these wounds lead to nasty infectious processes.

A dreaded postextraction complication is the so-called dry socket. This is a painful, localized osteitis, associated with the absence of an organized clot to protect the alveolar bone. The usual course of treatment ordinarily requires from two to three weeks. With the addition of local sulfonamide therapy, various clinicians have reported a reduction in healing time of 30 to 50 per cent.[10, 11]

An even greater field is the prevention of complications by the prophylactic local use of sulfonamides following an operation. For example, there was always a high expectancy of postoperative complications following the extraction of unerupted and impacted third molars. Surgical procedures of this nature involve a definite amount of trauma to both soft and bony structures and invite a reaction, first to the trauma itself, and second to the infection of the weakened tissues by mouth organisms. It is important to emphasize the reports, therefore, that postoperative pain, trismus, swelling, rise in temperature, and localized lymphadenopathy have been markedly reduced in the great majority of cases where sulfanilamide or sulfathiazole powder has been placed in the socket. Healing has been speeded, and potentially dangerous infections in the floor of the mouth, the submaxillary space, and along the fascial planes of the face and neck have been avoided.[10]

Another procedure whose prognosis has been greatly improved is the surgical removal of infected cysts from the jaws. It is now possible to enucleate the cyst, and, where teeth penetrate the cyst cavity, cut away all of the infected tooth apex and bone, and sterilize, fill, and seal off the root canals in which the infectious process originated. Then the clean bony cavity is powdered with sulfanilamide, and the mucous membrane flap is closed tight. The minimal nature of the postoperative reaction and the absence of reinfection result in healing by primary union, and eliminate weary weeks of irrigation and change of dressings and packings.

A solution or suspension of sulfonamide powder, when used for irrigation, has resulted in decreased pain and postoperative sequelae and has helped to prevent secondary infection in compound fractures, the surgical repair of cleft palate, and even when applied to various shallow ulcerations of the oral mucous membranes.

Toxic reactions from the local oral use of sulfonamides are not reported. There has been noted an increased tendency for oozing and secondary hemorrhage from some wounds thus treated. This is an undesirable feature, but not serious. Several investigators have reported an increase in the healing time of sockets with the local use of the sulfa drugs. This is possibly attributable to a technique which violates a surgical principle. For, if a compressed tablet or cone of sulfanilamide is inserted into a socket, or if the socket is packed full of powder and is allowed to remain undisturbed, the drug will act as a foreign body, and healing will be delayed until it is absorbed or thrown off. Enough finely powdered drug should be used just to coat the surface of a wound that is to be closed. The granules should be as fine as possible to ensure their absorption.

An attempt is now being made to incorporate sulfonamide therapy into our techniques for filling root canals and for capping vital pulps. First, a series of experiments was performed* to help establish in vitro the rationale of our clinical procedures. A pure strain of *Streptococcus viridans alpha* was obtained by culturing the apex of a random infected tooth after extraction. Melted agar was inoculated with this organism, blood added, and a series of identical pour plates prepared. As soon as the gel was hard enough to permit handling, 1 cm. squares of different combinations of drugs were placed on the surface. After twenty-four to thirty hours' incubation, the plates were examined. Results are noted in Table I.

The degree of inhibition of bacterial growth ranged from zero to four plus, and was gauged by the size of the circle of nongrowth surrounding the drug.

TABLE I

SUBSTANCES USED	INHIBITION
1. Zinc oxide and eugenol paste (ZOE)	++
2. ZOE plus talc	++
3. ZOE plus sulfanilamide	+++
4. ZOE plus sulfathiazole	++++
5. ZOE plus ammoniacal silver nitrate	++
6. ZOE plus ammoniacal silver nitrate plus sulfanilamile	++++
7. ZOE plus ammoniacal silver nitrate plus sulfathiazole	++++
8. Control	0

*At the bacteriology laboratories of Mt. Sinai Hospital, New York, N. Y.

These results encouraged us to modify our root canal therapy. It has been reported in the literature[12] that hot sulfanilamide solutions for irrigating apical infections have been used favorably. We use a sulfanilamide paste as a dressing in root canals between treatments. We had been employing a soft paste of zinc oxide and eugenol plus a drop of ammoniacal silver nitrate to coat and medicate the walls of the canal and to serve as a cement film for the gutta percha points which constituted our final filling.[13] We now incorporate a small quantity of sulfanilamide powder into a thin mix of zinc oxide and eugenol and use this as our cement. Results seem favorable at this early date.

We have also performed a small selected series of pulp cappings in permanent posterior teeth. With a minimum of trauma, all caries is removed, and the vital, bleeding exposure uncovered. The cavity is gently dried and phenolized. Then a few fine grains of sulfanilamide are teased over the exposure, the cavities filled with zinc oxide-eugenol-sulfanilamide paste, and the occlusal stresses on the tooth are relieved. At subsequent visits, part of the filling is removed, an oxyphosphate cement base placed, and a permanent filling inserted. It is much too early to draw conclusions from so small a group of cases. At present, the teeth we have followed are still vital to the electric pulp tester. Further studies are indicated before this technique can be recommended for general application.

In conclusion, we find that the general principles which guide the physician and surgeon in their use of the sulfonamides are equally applicable to oral and maxillary wounds and infections. The sulfa drugs are an important addition to our armamentarium but must not be used to replace the fundamental rules of technique and surgery. They also offer promise as an adjunct in root canal therapy and in pulp capping.

We must know of the hazards inherent in their systemic use and must be extremely careful to watch for them and to recognize the danger signs early. The intelligent and controlled use of the sulfonamides is a tremendous stride forward in our fight against disease.

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HEMORRHAGE

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SINCE dentists are to act as assistants to physicians in case of a bombing disaster, and in some cases be the only medical aid available, it is well at this time to consider the fundamentals of blood physiology, the results of loss of blood, the control and treatment of hemorrhage, and the replacement of the blood volume by fluids, by whole blood, or any of its parts, when indicated.

Not only must the dentist be familiar with the control and treatment of hemorrhage from the mouth and jaws, but he must be able to cope with hemorrhage anywhere in the body, should an emergency arise and he be the only available attendant.

Hemorrhage is the escape of blood from the enclosed blood circulatory system. The blood may escape into the body cavities, into the tissues, or into the open.

Bleeding may take place from the capillaries in the form of continuous oozing, from the venules and veins in the form of a continuous flow, and from the arterioles and arteries in the form of spurting.

Hemorrhage may be seen or may be concealed. The diagnosis of hemorrhage which may be seen is obvious and there are certain procedures by which it may be arrested. The diagnosis of concealed hemorrhage is more difficult and can be made only by diligent search for the signs and symptoms denoting the presence of such a condition.

Hemorrhage is primary when it occurs immediately following injury or immediately following an operation; it is intermediate when it occurs within twenty-four hours and is secondary when it occurs after twenty-four hours.

No method used to stop hemorrhage is of value if the necessary factors to produce coagulation are not present. Certain blood dyscrasias such as hemophilia, thrombocytopenic purpura, and leucemia have a defective coagulability or a disturbance of the permeability of the capillary endothelium.

Howell's[1] theory of normal coagulation, with slight modification, is still the accepted theory.

As explained by Kracke[2] normal coagulation is initiated by the escape of thromboplastin (cephalin) (thrombokinase) from incised or traumatized tissues, and from the platelets.

Antithrombin (heparin) existing in the blood, after being released from the liver, unites with the thromboplastin, which, in turn, releases prothrombin, the inactive precursor of thrombin.

Prothrombin is derived from the platelets and is said to be held in an inactive state by the antithrombin (heparin). Prothrombin is activated by calcium ions to form thrombin. Thrombin, in turn, unites with fibrinogen, which is said to exist in the blood plasma, to form fibrin, which is the first evidence of

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the coagulation process. The cellular elements of the blood become enmeshed in the fibrin, completing the clot. The completed clot plus platelets causes retraction with cessation of bleeding.

It is evident that a deficiency of fibrinogen, calcium, prothrombin, thromboplastin, or platelets, and an increase in the amount of antithrombin, or excessive permeability of the capillaries could be responsible for prolonged bleeding. Two important requisites preoperatively are a knowledge of the bleeding and clotting times of the individual.

DETERMINATION OF BLEEDING TIME

Bleeding time is the time required for the cessation of hemorrhage when blood forms from a deep puncture in the finger or lobe of the ear. The time is taken when the puncture is made, and every half minute the blood is blotted with filter paper. The interval between the first and the last drop is considered the bleeding time. If bleeding is longer than ten minutes, the bleeding time is prolonged. One to three minutes is the normal time. Although not entirely accurate, this method gives a clue to the possibility of the presence of some blood disturbance.

Essential thrombocytopenic purpura, aplastic anemia, and aleucemic leucemia cause a prolonged bleeding time. Even though sutures may be inserted to stop a hemorrhage, in these conditions bleeding will continue through the needle punctures. In thrombocytopenic purpura, not only are the platelets decreased but there is a marked permeability of the capillaries and oozing continues indefinitely.

DETERMINATION OF COAGULATING TIME

The time between the flow of blood at the site of the puncture and the formation of fibrin on a slide or watch glass is considered the coagulation time. The normal coagulation time varies from two to eight minutes.

Coagulation time can be determined by placing a few drops of blood on a glass slide. A needle is drawn through the blood at half-minute intervals. When a thread of fibrin can be picked up with the needle point, coagulation has begun.

Another simple method is to place a few drops of blood in a watch glass. An ordinary air rifle BB shot is placed in the blood and, at intervals, is allowed to roll through. When the shot has become enmeshed by the fibrin, coagulation has taken place.

Coagulation time is prolonged in hemophilia. The time varies from fifteen minutes to many hours. It is also prolonged when a hypoprothrombinemia exists.

Prothrombin is that factor in the blood stream which is converted into thrombin in the presence of calcium ions. It had been noted that when a vitamin K deficiency existed, the prothrombin level was low. As a result, there was impaired clotting power with prolonged blood coagulation time. It was further noted that unless vitamin K was carried through the intestinal mucosa by bile, the prothrombin level remained low. When a bile deficiency existed due to a damaged liver or bile obstruction, the percentage of prothrombin in the blood was below normal. The administration of vitamin K by itself did not raise the level, but when given with bile or choleic acid the prothrombin deficiency was corrected.[3]

There are four basic factors necessary for adequate prothrombin formation:[4]

1. A diet containing adequate vitamin K.
2. Presence of bile in the intestinal tract.
3. A normal absorptive intestinal surface.
4. A liver capable of effecting the conversion into prothrombin.

As far as hemorrhage is concerned unless one or more of the factors enumerated exist, the administration of vitamin K is of no practical value. It is of value only when a deficiency of prothrombin exists.

If it becomes necessary to consider the prothrombin clotting time preparatory to operation, there are a number of methods by which this may be determined. The normal prothrombin clotting time is about 20 seconds. If clotting takes more than 45 seconds, the patient is a potential bleeder.

PROFUSE BLEEDING WITH NORMAL BLEEDING AND COAGULATION TIMES

There are several conditions, nonthrombocytopenic in character, in which the bleeding, coagulation, and clot retraction times are normal, but bleeding is profuse and prolonged following accident or operation. These conditions are characterized by capillary dysfunction in which the capillary wall is in a temporary altered state. Terms used for this state are capillary weakness, weakened capillary resistance, or increased capillary fragility.

The clinical significance of such a condition is seen following the multiple extraction of teeth around which the mucous membranes have long been infected. There is a continual oozing which is difficult to control and which can be stopped only by drastic measures.

TOURNIQUET (RUMPEL-LEED) TEST

A tourniquet test for capillary resistance is made by placing the cuff of a blood pressure apparatus on the arm of the patient and holding it at the diastolic pressure for five minutes. If petechiae appear, the test is positive. If a blood pressure apparatus is not available, any tourniquet medium will do.

Certain skin conditions in the erythema multiforme group, Henoch's purpura, Schönlein's purpura, and allergic states such as urticaria and angioneurotic edema manifest this capillary weakness.

EMERGENCY TREATMENT OF HEMORRHAGE

It is evident, therefore, that hemorrhage may be traumatic or pathologic. In an emergency, when immediate cessation of hemorrhage may be the difference between life and death, there is no time for tests of any kind. The hemorrhage must be stopped, the blood circulatory volume restored, if indicated, and shock, when concurrent, must be combated.

To stop hemorrhage in an emergency, several factors must be considered. Is the bleeding from the head, trunk, or extremities? How much blood has been lost? Is the victim in shock? Is he conscious?

Injuries to the scalp are apt to result in profuse hemorrhage. The lateral and posterior aspects are supplied by the temporal, the posterior auricular, and

the occipital branches of the external carotid artery, while the anterior part is supplied by the frontal and supraorbital branches of the ophthalmic artery. The veins accompanying the two arteries empty into the ophthalmic vein; this in turn drains into the cavernous sinus.

Single limited wounds can be controlled by pressure over the bleeding area, if kept up long enough. Hemorrhage from extensive or multiple wounds of the scalp is troublesome. First aid consists in the application of a rubber tourniquet wrapped around the head until the patient has arrived at a first-aid station or hospital where the bleeding points can be clamped and ligated. In using hemostats for clamping bleeding vessels, only the bleeding point should be secured. The grasping and ligation of excessive tissue result in necrosis with sloughing.

Bleeding from the lower external nose can be stopped by pressure in the nasolabial fold over the lateralis nasi vessels. Bleeding from the upper external nose can be stopped as a first-aid measure by pressure over the supraorbital and infraorbital foramina. Hemorrhage from the internal nose may occur from the septal mucosa, the lateral nasal mucosa, or the mucous membrane covering the turbinates. If the bleeding is from the anterior two-thirds of the nose, it may be controlled by squeezing the alae against the septum with steady pressure for about ten minutes. Should severe hemorrhage be encountered, the nasal cavity must be packed. Occasionally, it may be necessary to pack the posterior nasal fossa to prevent the blood from entering the esophagus or larynx. This is accomplished in the following manner:

"A soft rubber catheter is passed along the floor of the nose until it appears below the soft palate. This end is seized with forceps and brought out through the mouth. A suitably sized sterile sponge around which a long piece of tape has been tied is prepared and the tape tied to the catheter. The catheter is withdrawn from the nose and the sponge pulled into the posterior nasal fossa. The nasal cavity can now be packed with sufficient bulk to exert pressure in all directions. The packing should not be left in place longer than twenty-four to thirty-six hours, lest infection result."[5]

Severe hemorrhage from the lips may be due to severance of the superior or inferior labial arteries which are branches of the external maxillary artery. The venous supply drains into the anterior facial vein which has no valves and communicates freely with the intracranial channels. The angular and supraorbital veins communicate with the ophthalmic vein, a tributary of the cavernous sinus. Some of the facial veins communicate through the pterygoid plexus with the cavernous sinus by tributaries which pass through the foramen ovale and foramen lacerum (medium). In hemorrhage from contaminated wounds of the anterior part of the face and lips, the quicker the venous supply can be shut off the less the chances are for a possible cavernous sinus thrombosis.

Arterial hemorrhage from the lips and anterior face can be stopped by pressure on the external maxillary artery as it passes over the facial notch anterior to the angle of the mandible. If the hemorrhage is from the lip only, the lip can be grasped between the fingers at the corner of the mouth and sufficient pressure exerted to stop the hemorrhage until two rubber tubes have been

placed on the beaks of a hemostat, and this clamped on the corner of the lip in place of the fingers. When the patient has been brought to a hospital and the hemorrhage has not ceased on the removal of the clamp, ligation can be performed.

HEMORRHAGE FROM EXTRACTION OF TEETH

Hemorrhage following the extraction of teeth may, at times, be troublesome. This may occur in patients with normal bleeding and clotting times and who do not suffer from any blood dyscrasia. Prolonged primary bleeding following extraction with a local anesthetic is rare, due to the presence of a vasoconstricting agent in the anesthetic. Profuse bleeding may take place from one of three places in a socket and is usually intermediate or secondary. Intermediate or secondary bleeding may occur:

(1) From the apex of the socket,
(2) From the bony walls of the socket, or
(3) From the mucous membranes.

The first requisite in treatment of bleeding is a clean field for visibility. If a suction apparatus is on hand, mucus, saliva and blood can be cleared away and the bleeding point or points determined. If a suction apparatus is not present, block or infiltration anesthesia is given and the area cleansed with sterile gauze. Many bleeding sockets can be controlled by this infiltration of a local anesthetic until a thorough examination can be made.

When a larger than normal apical arteriole or venule has been severed as a result of the extraction, the blood will be seen to well up from the apex. A sterile gauze strip is packed into the socket and a sponge is placed over it with pressure exerted into the socket by the opposing teeth. The sponge can be removed in two hours and the gauze strip in twenty-four hours. If necessary, a Barton type of bandage may be applied to help keep continuous pressure within the socket.

Examination of dental x-ray films of the mandible sometimes reveals rarefied linear radiolucencies consistent with dilated nutrient canals. At times, these may be seen to reach the ridge of the alveolus through the socket wall. Bleeding may be profuse and sustained in these conditions, and occasionally spurting or oozing occurs at the crest of the alveolus. Such hemorrhage from bone may be controlled by placing a blunt instrument over the bleeding spot and tapping it with a mallet. The crushing of the bone causes cessation of the hemorrhage.

If the bleeding is from the bony socket wall, drying the socket and rubbing Horseley's bone wax with an amalgam spatula into the bone will control the hemorrhage.

If, during the extraction of a maxillary molar, the posterior palatine mucous membrane is deeply lacerated with resulting hemorrhage, it can be controlled by pressure over the greater palatine foramen or by passing a suture in the mesiolateral direction through the palatal mucosa distal to the bleeding area if it is arterial. If the bleeding is venous, a suture is passed mesially to the bleeding gums.

Following multiple extractions in the presence of infected gingivae, bleeding may be profuse and continuous. This may be due to weak capillary walls which may be eroded. As long as pressure is exerted over the gums, bleeding ceases, but as soon as pressure is released, it starts again.

The use of hemostatic agents to control postextraction hemorrhage should be considered here. These agents may be classed as vasoconstrictors, astringents, animal serums and individual blood elements.

The *vasoconstrictors* are epinephrine (adrenalin), synephrin, neosynephrin, and ephedrine. Of these, adrenalin 1:10,000 is effective during operative procedure to keep a clean field when there is continuous oozing. A gauze strip saturated with adrenalin and held for a moment with pressure over the bleeding area will control the bleeding temporarily. When vasoconstrictors are used locally after extraction, primary constriction is followed by secondary dilatation with much more profuse bleeding.

The more commonly used *astringents* are alum (aluminum and potassium sulfate), antipyrine, tannic acid, Monsel's solution (liq. ferri subsulphatis—U.S.P.), and peroxide of hydrogen.

Alum will control oozing from superficial lacerations of the oral mucosa. A mixture of *antipyrine* and *tannic acid* powders put on gauze which is inserted into a bleeding socket over which pressure can be exerted is at times an excellent agent. For oozing mucous membranes, very cold *glycerite of tannic acid* is valuable. *Monsel's solution,* although an effective styptic, forms a hard black mass and is disagreeable to use. *Hydrogen peroxide* will arrest gingival oozing but should not be used in bone. This agent gives off nascent oxygen when in contact with organic material and if infected bone is present, infection may be pushed by the gas into the uninvolved bone trabeculae.

Animal serums and individual blood elements may be used topically or hypodermically. When used hypodermically, a dermal sensitivity test should be made. Coagulose, hemoplastin, thromboplastin, and rabbit thrombin are some of the agents which may be used.

Patients with a history of profuse bleeding following extractions may be given 150 c.c. of plasma, prophylactically, and have the sockets packed with dried plasma. Dameshek[6] reports that "almost complete rehabilitation of one patient with severe hemophilia was accomplished by the injection of 125 c.c. of plasma given weekly for three months."

In emergency, hemorrhage following multiple tooth extractions, with marked bleeding of the gingival tissues, can be controlled by animal serum such as 1,500 units of diphtheria antitoxin.

Dameshek also reports the use of rabbit thrombin as a local hemostatic agent. Mallett[7] studying the use of thrombin as a local hemostatic is enthusiastic with its results.

My experience with thrombin, although not large at this time, leads me to believe that it has an effective place among the agents used for hemostasis.

CONTROL OF HEMORRHAGE FROM THE EXTREMITIES

The dentist, through his training, is better able to cope with hemorrhage from the face and mouth than from other parts of the body. Nevertheless, in

these days of possible disaster, he should be familiar with the methods used to control hemorrhage from the extremities.

Hemorrhage from the upper extremity can be controlled either by pressing the brachial artery against the humerus, or by a tourniquet. When the latter is used, it must be loosened every twenty minutes in order that the complications due to the shutting off of the blood supply may be avoided. Ligation of the bleeding points should be made as soon as possible.

Hemorrhage from the lower extremity can be controlled by compression of the femoral artery immediately below Poupart's ligament. The artery here is superficial and can be compressed against the ascending ramus of the pubic bone. The femoral artery can also be compressed by a tourniquet placed over the middle third of the thigh.

BLOOD TRANSFUSION

The problems following the serious loss of a large volume of blood must be met with skill and judgment. With the loss of much blood, there is a decrease in the circulating blood volume. The blood pressure falls, there is cerebral anemia, and the face is pale with cold sweat on the forehead. The pulse is weak and thready. The respiratory symptoms are those of anoxemia, since the loss of blood is associated with decreased red blood cells, and the oxygen-carrying power necessary to support respiration and the respiratory needs of the tissues is markedly lessened.

A progressive *decrease* in the volume of respiration indicates recovery. A progressive *increase* in the volume of respiration indicates that, without a transfusion of blood, the patient will die. There are certain measures by which nature attempts to compensate for the loss of blood to the heart and brain. These compensatory mechanisms are:

1. Vasoconstriction of the capillaries, venules, and arterioles.

2. Passage of tissue fluids back into the blood vessels because of reduction of the hydrostatic pressure upsetting the equilibrium of the forces. The normal fluid distribution in the body is maintained by means of a balance of two forces; hydrostatic pressure within the capillaries which tends to drive the fluid out of the vessels, and osmotic pressure of the proteins in the plasma which strives to retain it.

3. Augmented respiratory effort increases the venous return to the heart by increasing the normal pumping action of breathing.

In the meantime the corpuscular and fluid loss of the circulation must be made up if the patient is to survive. The solution used must restore the deficiency of the blood protein in order to hold the fluid within the vessels because of its osmotic pressure. The only way to restore volume protein loss and remove the anoxemia is a blood transfusion. Oxygen-carrying erythrocytes are supplied and the fluid volume is not temporary, such as follows the administration of normal saline or glucose solutions.

In civilian life, whole blood transfusions are more feasible than in actual warfare combat. During combat, preparations of human plasma or serum are available and can be quickly administered. Before whole blood can be used, diseases such as syphilis and malaria must be ruled out of the donor's blood.

Transfusion of fresh whole blood can be given directly or indirectly. The direct method requires operative skill, as the use of certain mechanical apparatus, requiring considerable training, must be used to transfer the unaltered blood from the donor to the recipient.

The indirect method is the one most widely used. It consists of the removal of the required amount of blood from the donor. When this is removed, it is mixed in a sterile receptacle with sufficient sodium citrate solution of 0.25 to 0.30 per cent to prevent coagulation. This mixture is injected into the vein of the recipient.

If fresh whole blood is not available, stored whole blood can be used. This is citrated or preserved blood which can be obtained from a so-called "blood bank." These "banks" are applicable for transfusions in the larger hospitals only, because of the technical problems and refrigeration necessary for the preservation of such blood.

If whole blood is not available, blood plasma has been shown to be equally effective for replenishing the protein content or restoring the fluid volume of the blood stream. Plasma can be liquid or dried. In shock, it is more effective than whole blood since shock is associated with hemoconcentration. Dried plasma powder is dissolved in distilled water and injected intravenously.

Transfusion with whole blood necessitates compatibility tests in order to avoid an untoward reaction through the use of mismated blood. All bloods can be classified into four groups based upon the distribution of two specific antigens in the red cells and two antibodies in the serum. The antigens in the red cells are called isoagglutinogens A and B, and the antibodies in the serum are called isoagglutinins a and b.

The recommended nomenclature is the Landsteiner classification which is as follows:

GROUP	AGGLUTINOGEN	AGGLUTININ
AB	A and B	None
A	A	b
B	B	a
O	None	a and b

Because group AB contains no agglutinins in the serum by which the donor's cells could be affected, the AB group is known as the "universal recipient." Because group O contains no agglutinogens in its red cells which the recipient's serum could agglutinate, group O is called the "universal donor." This, however, is not really true, because there are numerous subgroups which may eventuate in post-transfusion reactions.

At present, agglutinogens A_1 and A_2 are recognized as occurring in connection with groups AB and A, giving rise to subgroups A_1, A_2, A_1B and A_2B.[8] The most recently discovered agglutinable factor in red cells is the Rh factor. Anti-Rh agglutinins formed after repeated transfusions may result in severe and even fatal reaction due to intragroup incompatibility.

CONCLUSION

Hemorrhage and shock are the two primary factors which will have to be dealt with by those associated with medical defense units should a disaster oc-

cur. Dentists who will be components of such units must familiarize themselves with the physiology, pathology, and treatment of these conditions, if mortalities are to be kept low.

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DENTAL DEVELOPMENT IN CONGENITAL SYPHILIS

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A CRITICAL analysis of the dental manifestations in congenital syphilis is timely in view of the recent educational, experimental, and clinical efforts to reduce the incidence of this disease. The purpose of this investigation was to determine the characteristic clinical, roentgenographic, and histologic dental changes and to consider their frequency, specificity, and pathogenesis in congenital syphilis. A further purpose was to correlate the observations on the deciduous and on the permanent teeth. Many writers have taken exception to the opinions expressed by Hutchinson[1] since his original description of the specificity of the abnormalities in the upper permanent central incisors in congenital syphilis.

REVIEW OF THE LITERATURE

One of the earliest references to dental alterations in syphilis was that by Sanchez[2] in 1785. Hutchinson[1] in 1858 gave the first complete report and described several disturbances in dental development which he thought were due to congenital syphilis. Subsequently, however, he modified his view and conceded that dental dystrophies other than the Hutchinson incisor could be caused by other factors. According to Magitot[2] (1881), eclampsia was the important etiologic factor of hypoplasia of the enamel. In 1884 the abnormal state of the permanent first molar in congenital syphilis was described by Fournier[3] and by Moon.[4] Cavallaro's report[5] in 1908 was an extensive clinical and histologic work. He and Stein[6] stated the belief that all types of dental hypoplasia are of syphilitic origin. Karnosh[7] and others[8] stated that the Hutchinson incisor is due to a deformity in the developmental lobes and is not an effect of transitory calcium deficiency (table). Extensive bibliographies are found in the reviews by Kranz[9] and Lebourg.[10]

MATERIAL AND METHOD

Material.—The present study was based on examinations of 133 consecutive patients in the children's syphilis clinic, Central Free Dispensary, Rush Medical College, the University of Chicago. In addition to children for whom a positive diagnosis of congenital syphilis had been made, there were others who so far had shown no signs of the condition. The latter children were born of syphilitic mothers (treated and untreated) and were being observed over a long period. Many of them were siblings of the children with congenital syphilis. There were also a few patients with acquired syphilis in the group.

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HISTORICAL REVIEW OF DENTAL STUDIES IN CONGENITAL SYPHILIS

AUTHOR	DATE OF PUBLICATION	OBSERVATIONS AND CONTRIBUTIONS
Sanchez	1785	Premature caries (black decay of teeth)
Hutchinson	1858	Notched and tapered permanent upper central incisors as part of Hutchinson's triad
Fournier; Moon	1884	"Mulberry molar," constricted and irregular occlusal surface of permanent first molar
Cavallaro	1908	Extensive histologic and clinical studies; did not distinguish between changes in form of tooth and enamel formation and believed all were caused by congenital syphilis
Stokes and Gardner*	1923	Roentgenographic diagnosis of unerupted Hutchinson incisors
Pfluger	1924	"Bud molar," constricted but smooth occlusal surface of first permanent molar
Karnosh	1926	Tooth-forming organs not seriously impaired in utero but suddenly at birth; not a transitory calcium deficiency but a deformity of developmental lobes
de Jonge-Cohen	1932	Hutchinson tooth has aplasia of medial edge tubercle and convergence of both approximal crown surfaces
Sarnat, Schour and Heupel	1941	Roentgenographic diagnosis of unerupted Hutchinson incisors and mulberry molars plus disturbance in enamel formation due to systemic disease at a later time
Sarnat and Shaw (this report)	----	Correlation of dental development with conditions found in deciduous and permanent teeth

*Stokes, J. H., and Gardner, B. S.: J. A. M. A. **80**: 28, 1923.

Method.—Medical History and Physical Examination: In every instance a medical history was obtained and a physical examination performed. In selected cases roentgenograms of the long bones were taken. The blood serum of every patient and the spinal fluid of some were tested for syphilis.

Dental Analyses: The dental examination, performed without knowledge of the patient's medical diagnosis, consisted of:

Gross Examination With Dental Explorer and Mouth Mirror: A record was made of the particular teeth which varied morphologically from the normal and those which showed evidence of disturbed enamed formation in the form of hypoplasia of the enamel (chronologic enamel aplasia).

Roentgenograms: These were taken routinely of every patient whose permanent teeth were not yet erupted or whose erupted permanent teeth showed abnormal form. The roentgenograms provided a graphic record of the enamel, dentine and dentinoenamel junction of the various erupted and unerupted teeth and permitted a more complete study of the presence or absence of teeth. Roentgenograms were also taken of selected extracted teeth from normal patients and patients with congenital syphilis.

Photographs: Black and white and colored photographs of the teeth were taken of a selected group of patients for permanent record. In addition, photographs were taken of selected extracted teeth from normal patients and patients with congenital syphilis.

Impressions and Models: Hydrocolloid impressions of the dentures were taken in several instances. Models developed from the impressions served as valuable records.

Histologic Material: Midsagittal ground sections were made in the mesiodistal plane of selected extracted teeth. These were obtained from patients with and without congenital syphilis.

BRIEF SUMMARY OF DENTAL DEVELOPMENT

Since the dental effects in congenital syphilis are of developmental nature, they can be best understood if the important stages in dental development are first reviewed. These are: (1) growth, (2) calcification, (3) eruption and (4) attrition. For this purpose, only the first stage (growth) of the deciduous and permanent central incisors, which are comparable to the other deciduous and permanent teeth, will be considered. This stage can be further subdivided into initiation, proliferation, morphodifferentiation, histodifferentiation, and apposition. Because the deciduous teeth develop earlier than the permanent teeth, they are in a later stage of development at any particular time in the life of the fetus or infant (Fig. 1). Consequently, systemic disturbances will produce a different effect on growing deciduous teeth than on growing permanent teeth.

Initiation (Deciduous Incisor).—Between the sixth and seventh weeks of intrauterine life an inward proliferation of the oral epithelium (*O. ep.*) begins (Fig. 1 *A*). This is the dental anlage (*D. a.*).

Proliferation (Deciduous Incisor).—Cellular proliferation continues until an invagination takes place and the connective tissue which borders the invaginated epithelial portion condenses and forms the dental papilla (*D. p.*), the primordium of the dentine and pulp (Fig. 1 *B*).

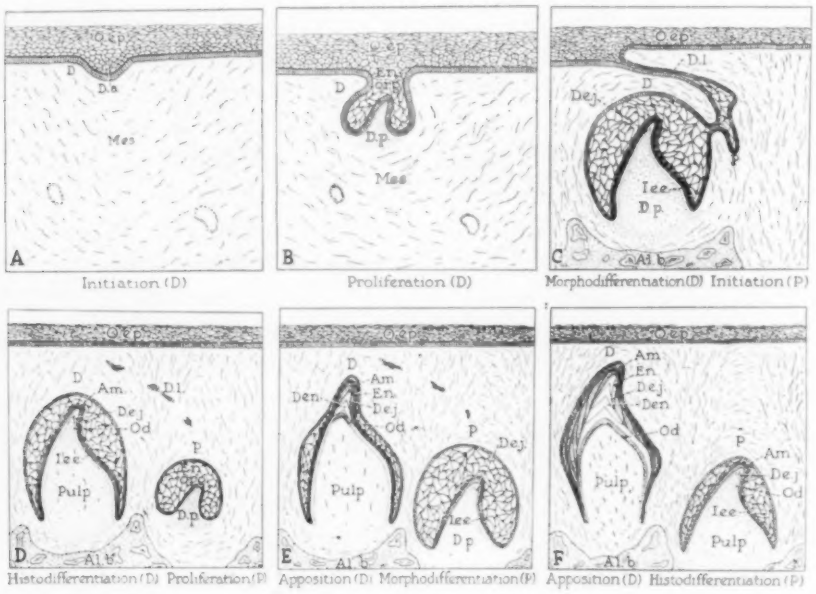

Fig. 1.—Diagrammatic representation of the development of the lower deciduous and permanent central incisors, showing: *D*, a deciduous tooth; *P*, a permanent tooth; *O.ep.*, oral epithelium; *D.a.*, dental anlage; *Mes.*, undifferentiated mesenchyme; *D.p.*, dental papilla; *En.Org.*, enamel organ; *I.e.e.*, inner enamel epithelium; *Dej.*, dentinoenamel junction; *D.l.*, dental lamina; *Den.*, dentine; *En.*, enamel; *Am.*, ameloblasts; *Od.*, odontoblasts, and *Al.b.*, alveolar bone.

Differentiation (Deciduous Incisor).—The enamel organ (*En. Org.*), which originates from the invaginated epithelium, gives rise to the inner enamel epithelium (*I. e. e.*). Two changes occur at this time: 1. Morphodifferentiation, the determination and differentiation of the dentinoenamel junction between the inner enamel epithelium and the adjacent connective tissue cells of the

dental papilla (Fig. 1 *C*). The dentinoenamel junction determines the basic pattern of the tooth. At about the time of this change (approximately the fourth month in utero), the initiation of the permanent incisors begins. 2. Histodifferentiation, the differentiation of ameloblasts (*Am.*) (enamel-forming cells) from the inner enamel epithelium, and of odontoblasts (*Od.*) from the adjacent connective tissue of the dental papilla (Fig. 1 *D*). This stage occurs about the fifth month in utero. The permanent tooth bud is undergoing proliferation.

Formation or Apposition of Enamel and Dentine (Deciduous Incisor).—The next stage is the formation of a cusp of dentine followed by a corresponding amount of enamel on the outer portion of the dentinoenamel junction. When this formation has been initiated, there is a synchronous recession of the ameloblasts and odontoblasts from each other. Incremental layers of enamel are apposed one on top of the other until the cusp is fully formed. Subsequent layers are apposed at the sides until the crown is complete. The enamel is thickest at the incisal edge; it gradually tapers off and ends at the junction of the anatomic crown and root. For each layer of enamel that is apposed, outside the previous one, a corresponding layer of dentine is apposed, within the previous one. After formation of enamel has ceased, formation of dentine still continues to complete the root.

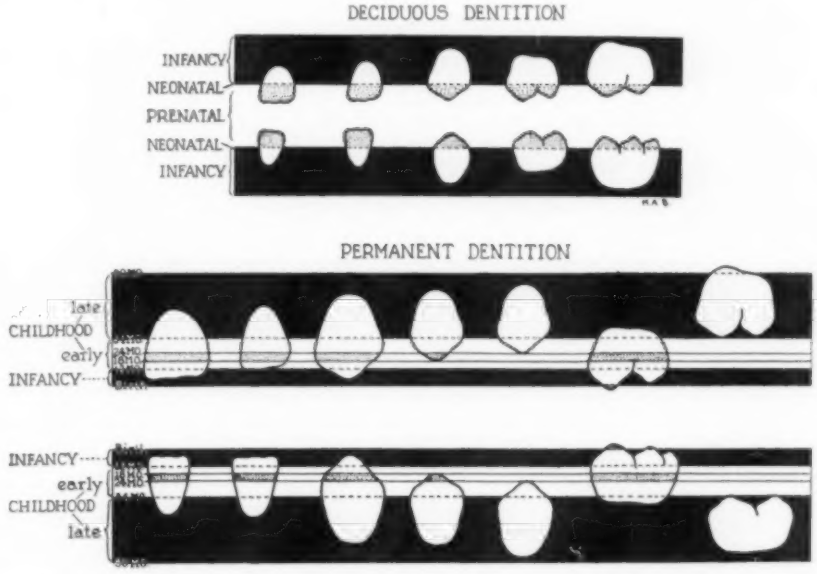

Fig. 2.—Diagrammatic representation of the chronologic development of the crowns of the deciduous and permanent teeth (from Sarnat and Schour[10b]).

Comment.—This same developmental process, with certain variations, is initiated at definite intervals for the various teeth in both dentitions (Fig. 2). At the time of birth the first molars and incisors of the permanent dentition (except the upper lateral incisors) are in the stage of morphodifferentiation, while the deciduous teeth are forming enamel (Fig. 1 *E*). The occurrence of a sufficiently severe systemic disturbance during development of the teeth may affect the particular stage in process at the time[11] and be expressed at a later date as one of the following dental dystrophies:

Stages in Growth	Dental Aberration
Initiation	Anodontia
Proliferation	Odontoma
Morphodifferentiation	Hutchinson incisor, Moon molar, peg-shaped teeth
Apposition	Hypoplasia of enamel (chronologic enamel aplasia)

Thus in congenital syphilis, because the form of the tooth is altered (morphodifferentiation), the primary requisite of the upper permanent central incisor and of the first permanent molar is a convergence of the lateral surfaces, with a resulting narrowed mesiodistal diameter of the crown (Fig. 3).

OBSERVATIONS

Of the 133 patients examined there were 3 children with acquired syphilis and 17 who were classified as nonsyphilitic because they were born of mothers adequately treated during pregnancy and because they had shown no signs of congenital syphilis. Forty others who had shown no signs of congenital syphilis were under clinical observation because they were born of syphilitic mothers who had inadequate or no treatment during pregnancy. Finally, there were 73 children who had a positive diagnosis of congenital syphilis and whose serum had reacted positively at some time.

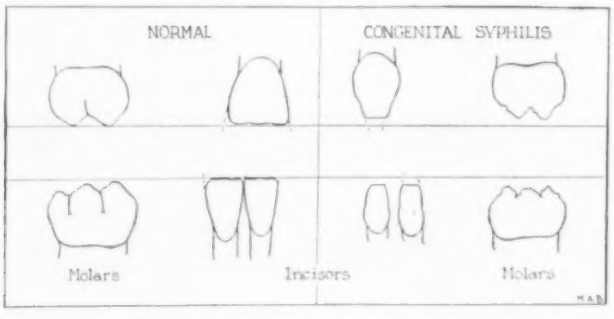

Fig. 3.—Diagrammatic representation of the gross morphologic characteristics observed in the teeth of a patient with congenital syphilis, compared with those of a normal control subject (from Sarnat, Schour and Heupel[18]).

Of the 73 children with congenital syphilis, there were 57 children 7 years of age or older whose permanent upper central incisors and first molars were erupted and examined clinically. Eighteen (32 per cent) had teeth which appeared to be characteristic of congenital syphilis. In a dental roentgen examination of the remaining 16 children with an unerupted permanent dentition, teeth with converging proximal surfaces, characteristic of congenital syphilis, were found in 4 patients (25 per cent). It is significant that in the groups of children whose condition was not diagnosed as congenital syphilis no child showed these characteristic dental changes.

Deciduous Teeth.—In the 4 patients who still had their deciduous teeth, there was hypoplasia of the enamel. The time of disturbed formation of enamel corresponded approximately with the neonatal period and the period of earliest infancy. The teeth, especially the central and lateral incisors, were badly worn and showed extreme caries. No disturbance in the morphologic pattern was seen.

Permanent Teeth.—*Incisors.*—Gross Observations: Two groups of permanent teeth were found to have a convergence of the proximal (lateral) sur-

faces, namely the incisors and the first molars. The teeth most frequently affected were the upper central incisors, usually both but sometimes only one. The morphologic pattern was extremely variable, and in several instances in which the distinction was not pronounced the teeth were considered normal. Twenty-two patients had Hutchinson incisors. The teeth of 14 of these patients had, in addition to a convergence of the lateral surfaces (Fig. 4 *A*), a midincisal notch (Fig. 4 *B*). The notch varied from slight to deep. The lower central and lateral incisors also showed a convergence of the lateral surfaces and an incisal notch, or the former alone, but not so frequently as the upper incisors (Fig. 4 *A* and *B*). The upper lateral incisor was conspicuous in that in only one instance did its pattern vary from the normal. In some patients malocclusion and a diastema between the two upper permanent central incisors were found.

Roentgenographic Observations: A pronounced convergence in the mesiodistal diameter of the dentinoenamel junction of the affected teeth (up to about the midcoronal level) was seen in the examination of the roentgenograms (Fig. 5 *A* and *B*). In addition, the notched defect of the midregion of the incisal edge could readily be seen. The thickness of the enamel appeared to be normal. Examination of the unerupted teeth in the roentgenograms revealed characteristic Hutchinson incisors in every instance in which the deciduous teeth had chronologic enamel aplasia (Fig. 6 *A* and *B*).

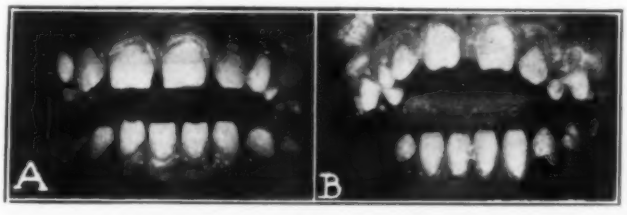

Fig. 4.—Intraoral photographs of patients with congenital syphilis and Hutchinson incisors. *A*, note in the upper central incisors the convergence of the proximal surfaces, the slight notch on the incisal edge and the diastema. This is the so-called screwdriver tyre. In the lower incisors, in addition to a convergence of the proximal surfaces, the notch on the incisal edge is more apparent. (See Fig. 5*A*.) *B*, note in the upper central incisors the convergence of the proximal surfaces in the incisal third of the crown, the distinct notch on the incisal edge and the diastema. In the lower incisors the convergence of the proximal surfaces is apparent; the notch on the incisal edge is not pronounced. (See Fig. 5*B*.)

Congenital absence of teeth (upper permanent lateral incisors) was noted in only 1 of 73 patients whose serum reacted positively for syphilis. This was confirmed by both gross and roentgen examinations.

Molars.—Gross Observations: The permanent first molars were not so consistently affected as were the central incisors. Here too, as in the incisors, the proximal surfaces were constricted so that approximately the occlusal third was narrower than normal. In addition, in some molars hypoplasia of enamel (chronologic enamel aplasia) was superposed. Of the 88 possible molars in the 22 patients with Hutchinson teeth, only 11 were diagnostic (6 patients); 27 were either absent or so carious that they were of no diagnostic value; 40 appeared to be normal, and 10 had hypoplasia of enamel but no distortion of the crown.

Roentgenographic Observations: Roentgenographic examination revealed the convergence of the sides of the molars and irregular enamel in certain

instances. This was also diagnosed prior to eruption (Fig. 6 *A* and *B*). It is interesting to note that the occlusal surfaces after eruption were subject to severe wear. This was demonstrated roentgenographically in the appearance of the molars prior and subsequent to occlusal wear.

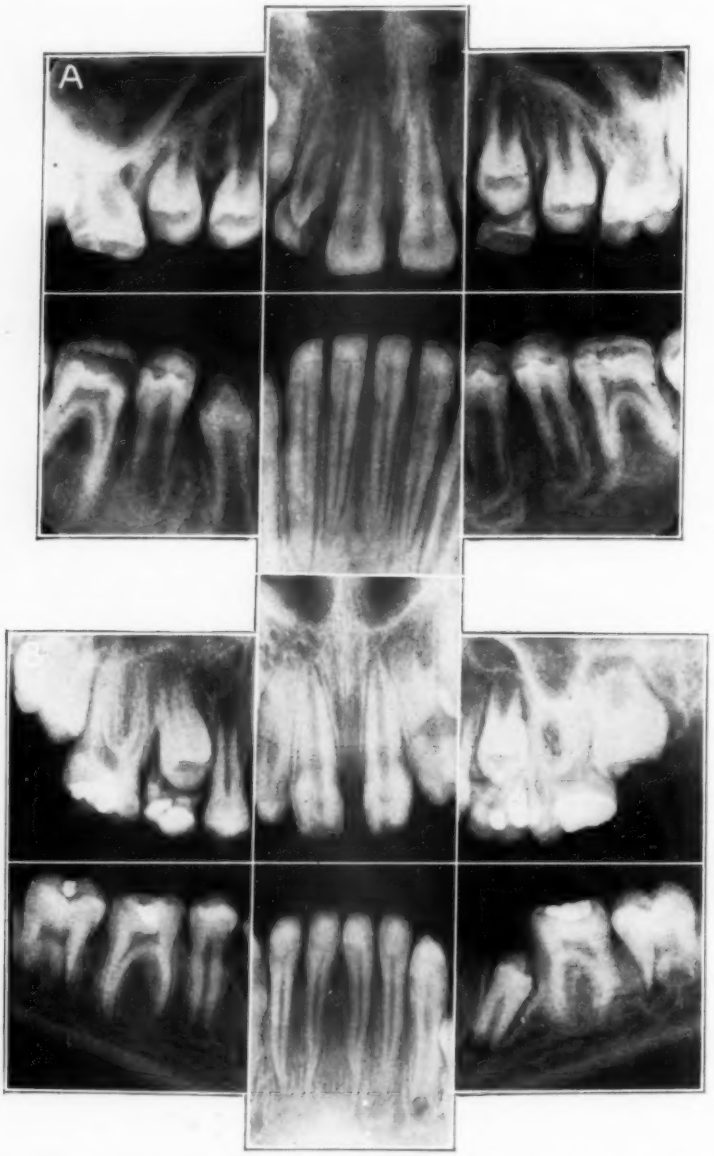

Fig. 5.—Intraoral roentgenograms of the incisor and molar areas of the same patients with congenital syphilis described in Fig. 4. Note the size and shape of some of the permanent incisors (except the upper lateral incisors) and the permanent first molars. (See Fig. 4.)

Histologic Observations: A comparison of the ground sections of Hutchinson and of normal incisors revealed differences in the size and form of the dentinoenamel junctions but no significant structural differences in the enamel and dentine. The dentinoenamel junction was smaller and constricted in about the incisal third of the crown (Fig. 7). Similar conditions were found in the molars (Fig. 8).

COMMENT

Systemic Disease and Growing Teeth.—Recent clinical studies[11a, b] have demonstrated the growing tooth to be an accurate permanent chronologic recorder of metabolic disturbances. The various stages of dental development occur in sequence at a definite time for each tooth. With the knowledge of the chronology of development, the time of the systemic disturbance, but not the etiologic factor, can be determined. The amount of dental development

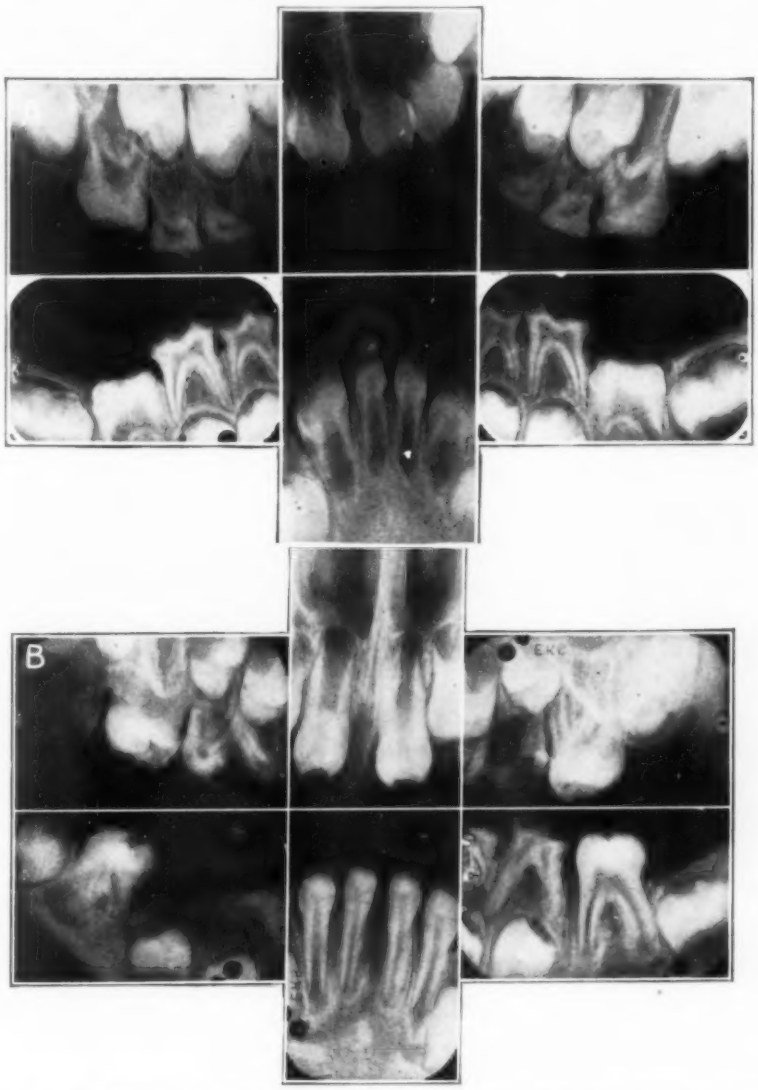

Fig. 6.—Intraoral roentgenograms of patients with congenital syphilis, showing unerupted permanent central incisors and first molars. Note the convergence of the proximal surfaces and the smaller size of the teeth. *A*, the permanent central incisors are screwdriver-like; the permanent first molars are mulberry-like. Note the irregularity of the incisal third and the occlusal surface. *B*, note the notch in the unerupted permanent upper central incisors. The permanent first molars are the bud form described by Pfluger.

at birth is dependent in part on the maturity of the infant. Thus a seven-month premature infant will not have as fully developed a dentition as a full-term newborn infant. The first permanent molars show evidence of tooth

formation and calcification at birth. The anterior teeth, except the upper lateral incisors, develop a little later and show no evidence of calcification until about the third month after birth (Fig. 2).

Fig. 7.—*A*, extracted Hutchinson incisors. Note convergence of proximal surfaces beginning at about the middle third of the crown, which gives it a screwdriver-like shape. *B*, mesiodistal ground section of a Hutchinson incisor. The significant histologic abnormality is the form of the dentinoenamel junction. *C*, mesiodistal ground section of a normal incisor. Compare the dentinoenamel junction with that in *B*. *D*, extracted normal incisors. Note the divergence of the proximal surfaces. *E* and *F*, roentgenograms of ground sections of the Hutchinson (*E*) and normal (*F*) incisors.

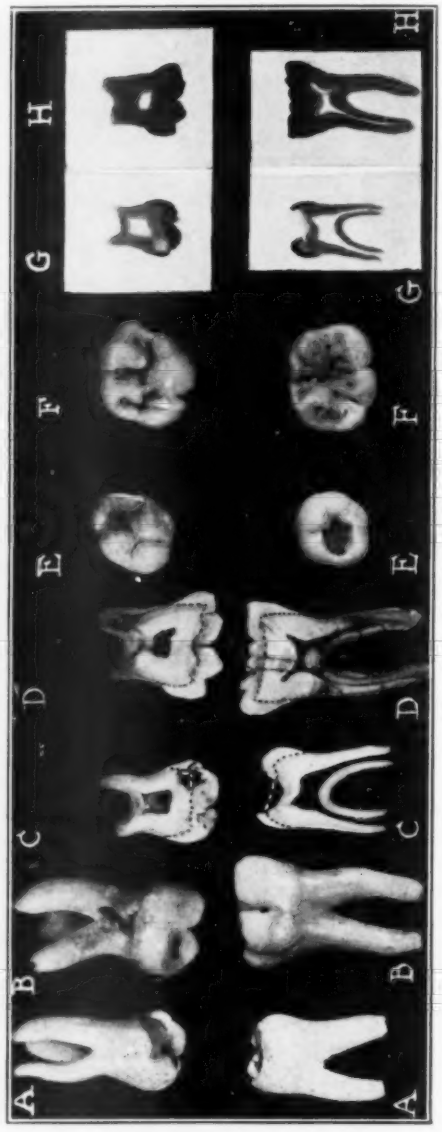

Fig. 8.—Extracted bud type and normal upper and lower first molars of second dentition. *A* and *B*, note difference in size and form in the buccal view of bud molars (*A*) and normal molars (*B*). *C* and *D*, mesiodistal ground sections of bud molars (*C*) and normal molars (*D*). The significant difference is in the outline of the dentinoenamel junction. *E* and *F*, note difference in size and form in the occlusal view of bud molars (*E*) and normal molars (*F*). *G* and *H*, roentgenograms of ground sections of bud molars (*G*) and normal molars (*H*).

The effects of systemic disease on growing teeth have been reported.[11a, b] Schour[12] has demonstrated the neonatal line in the teeth in the process of development at the time of birth. The separation of the normal fetus from its placental circulation and the adjustments made during the neonatal period by the infant are severe enough to be recorded in the teeth. The infant who, in addition, has to contend with a syphilitic infection at that time may register the effects of this more severe disturbance in the portions of the deciduous and permanent teeth actively developing at that particular time.[13]

However, it should be pointed out that the clinical course of the infant and the dental conditions cannot always be correlated. Thus there were several

patients who had a history of florid syphilis but no characteristic changes in the teeth. Others had a history of a relatively undisturbed postnatal life but nevertheless had abnormal dental development. Consequently, the Hutchinson incisor and the bud or mulberry molar should not necessarily be considered indicative of the severity of the disease during the neonatal and infancy periods.

Deciduous Teeth.—In the patients studied, the anterior deciduous teeth had been shed or were badly worn. The posterior teeth showed aplasia of enamel characteristic of a disturbed development during the neonatal and earliest infancy periods. These observations were consistent with those of Karnosh.[7] No changes in the morphologic pattern were seen. This was explained on the basis that morphodifferentiation and the dentinoenamel junction were determined in utero, before any severe systemic disturbance affected the teeth (Fig. 1 *C*). The disturbance in enamel formation (hypoplasia of the enamel) was not pathognomonic of either congenital syphilis or any other disease.[11b]

Permanent Teeth.—Incisors: The normal permanent upper central incisors are subject to considerable morphologic variation. Generally, however, the crown has the form shown in Fig. 9 *A*. The three mamelons on the incisal edge are remnants of the developmental lobes. These are worn soon after the teeth are in occlusion (6 to 9 years).

The permanent upper central incisors in children with congenital syphilis begin to undergo morphodifferentiation at about birth (Fig. 1 *E*). The first evidence is the three lobes, the middle one of which may be more affected than the two lateral lobes. According to De Jonge-Cohen,[8b, c] there may be an aplasia of the median lobe. Concomitant with this, the lateral lobes may be smaller and closer together.[7] These factors produce a narrowed and constricted dentinoenamel junction. The extent of the deformity is dependent on the degree of aplasia of the central lobe and usually involves only the incisal third of the tooth. It is on this distorted dentinoenamel junction, which determines the future morphologic form of the tooth, that normal enamel is laid down.

Pitts[14] has classified the various types of Hutchinson teeth and has shown the difficulty which may be encountered in deciding whether a tooth has normal morphologic characteristics or not. Because the degree of convergence of the lateral surfaces may vary, the diagnosis may sometimes be questionable (Fig. 9 *B*). The primary requisite of the Hutchinson incisor is the convergence of the proximal surfaces, with a resulting narrowed mesiodistal incisal diameter of the crown (Fig. 9 *C*). In addition to the screwdriver-like effect of the tooth, a central notch may be present on the incisal surface (Fig. 9 *D*). Contrary to the views of some writers, this notch is present before the tooth appears in the oral cavity (Fig. 6 *B*). However, with occlusion, the tooth structure, which may be fragile in the central portion of the incisal region, may wear, and the notch may become accentuated.

The permanent lower central incisors may also appear morphologically similar to the permanent upper central incisors (Fig. 4), but the abnormal forms do not appear so frequently. The cuspids (canines) are seldom involved.

This is probably due to the fact that these teeth usually develop somewhat later. The permanent upper lateral incisors are not affected, because they do not begin development until a much later time (at about 10 months).

Molars: The permanent first molars have also been described as showing characteristic abnormalities in congenital syphilis.[15] The cusps are generally crowded together on a crown surface of dwarfed dimensions. In normal patients and patients with congenital syphilis the average mesiodistal diameter of the permanent first molar at the cuspal level was found to be 13.4 and 8.3 mm. respectively.[16] Karnosh[7] expressed the opinion that the incisor arose from a diminutive enamel organ but failed to attribute the molar to the same source. However, his illustrations clearly showed a dentinoenamel junction which was narrower than normal in the occlusal region. This narrowed dentinoenamel junction, as in the permanent central incisor, was produced at the time of morphodifferentiation. Subsequently enamel was apposed on this abnormal pattern. Pfluger[16] called this the bud form molar, because it gave the impression that the crown had not been fully outfolded (Fig. 6 *B* and 8 *A*). In addition, enamel formation was sometimes affected, and aplasia of the enamel occurred for the particular period (Fig. 6 *A*). This caused an irregular occlusal area, and for this reason the tooth had been called the mulberry molar (also Moon or Fournier molar).

Thus the teeth which are affected and may be of diagnostic value in congenital syphilis are the permanent upper central incisors, lower central and lateral incisors and first molars (Fig. 3).

Specificity and Differential Diagnosis.—Hutchinson[1] described dental conditions which he believed to be pathognomonic of congenital syphilis. His views, however, were questioned by Magitot,[2] Black,[17] and others.[2] It is important at this time to recall that in congenital syphilis the dentionenamel junction is dwarfed in the permanent upper central incisors and first molars. Consequently the crown is smaller. This should not be confused with hypoplasia of the enamel (chronologic enamel aplasia) (Fig. 10 *C*), which is a disturbance not in the outline of the dentinoenamel junction but in the enamel formation. Hypoplasia occurs at a later time and may be caused by rickets, hypoparathyroidism or fluorosis. The combination of the Hutchinson incisor and hypoplasia of the enamel, though rare, does occur (Fig. 6 *A*).[18] Pfluger[16] emphasized that in congenital syphilis one deals not with hypoplasia of the enamel but with the changed form of the entire tooth. If the hypoplasia occurred early, so that the enamel on the incisal edge was missing, the incisor would have a notched appearance (Fig. 10 *B*). The dentinoenamel junction, however, is not affected. In addition, not all Hutchinson incisors are notched (Fig. 4 *A*). For these reasons, the notched incisor alone should not be used as a means of diagnosis.

Congenital syphilis is unique as a systemic disease already present at the time of birth and often producing a severe and continued malnutrition in earliest infancy. This is an added disturbance, with which the newborn infant must cope. Thus morphodifferentiation of those permanent teeth active at the time may be affected, although the severity of the disease cannot always be correlated with the dental condition. Karnosh[7] stated that the tooth-forming

organs were not seriously impaired in utero and that most of the deciduous teeth as well as the prenatal caps of the first permanent molars were fairly well formed in spite of the presence of active syphilis in the fetus. The disease has a rather precipitous effect on the active enamel organ as soon as the infant is separated from the placental circulation.

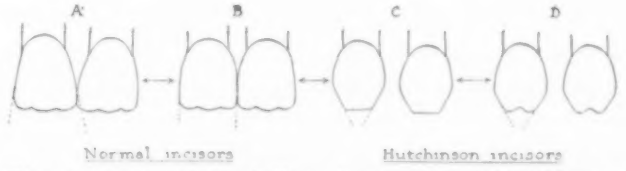

Fig. 9.—Diagrammatic representation of normal and Hutchinson incisors.

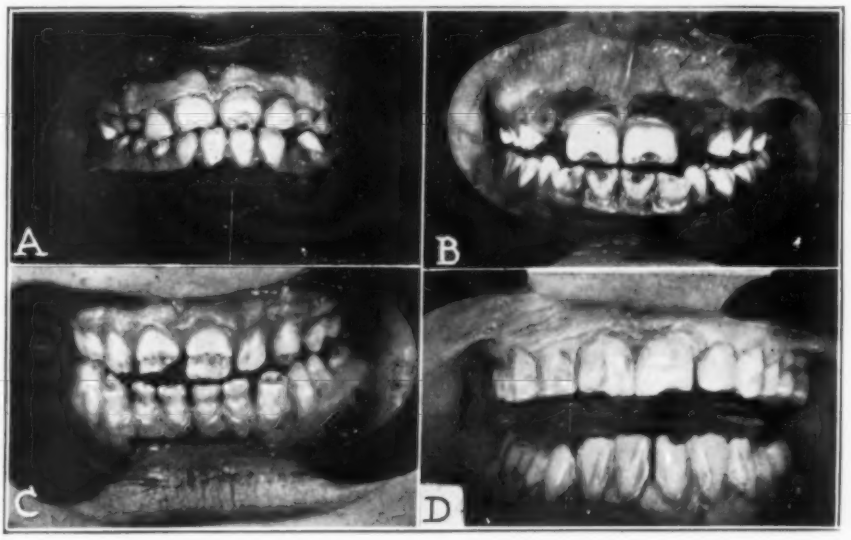

Fig. 10.—Differential diagnosis of the Hutchinson incisor. *A*, typical Hutchinson incisors, with convergence of the proximal surfaces and a notch of the midincisal edge. This was caused by a disturbance in dental development at about birth and shortly thereafter. *B*, notched incisors due to lack of enamel formation (hypoplasia of enamel). This abnormality was caused by a disturbance in dental development when the child was about 3 to 4 months of age. *C*, hypoplasia of the enamel due to a systemic disturbance between approximately 6 and 12 months of age. *D*, notched incisors due to environmental rather than developmental factors (abnormal local wear of teeth [abrasion] due to repeated holding of nails between teeth by an upholsterer). The only lesion characteristic of and due to congenital syphilis is that shown in *A*.

Black[17] did not question that the Hutchinson incisor was distinctly different from teeth with hypoplasia of enamel. He did believe, however, that any systemic disease which might be present at the same time might give the same result. Nevertheless, in a previous clinical study of dental dystrophies,[11b] the Hutchinson incisor or Moon molar was not demonstrated without the presence of congenital syphilis. Because congenital syphilis is active at the time of birth, teeth which are growing will be affected in the developmental processes proceeding at the time. Since no other comparable severe systemic disturbance which will cause the Hutchinson incisor is known at the present time, this tooth may be considered to be pathognomonic for congenital syphilis. However, it would be valuable to study the teeth of patients who had a severe disturbance, like tuberculosis, pyloric stenosis or diarrhea, during the neonatal

and infancy periods. Thus, Wolfe[19] and Schour and Kronfeld[20] have reported cases of hypoplasia of the enamel of the deciduous teeth due to rickets and an injury of the brain which occurred during the prenatal and neonatal periods, respectively. There was no history of syphilis. If the permanent teeth had a narrowed dentinoenamel junction, the effect of syphilis on the teeth would not be specific.

In partial anodontia, arachnodactylia, facial hemiatrophy, mongolian idiocy, achondroplasia and a few other conditions, the upper permanent incisors may have a narrowed dentinoenamel junction and a peg shape. Whereas the incisal third is affected in a Hutchinson incisor, the entire crown is affected and other teeth show similar disturbances, which cannot be correlated on a chronologic basis with events during the neonatal period and earliest infancy. With a little practice and experience, on examining the dentition of a child one can unmistakably determine whether it belongs to an idiot or to a child with hereditary syphilis.[5] Furthermore, these malformations should be distinguished from the semilunar notch frequently seen in the incisors of upholsterers and carpenters, who keep nails between their teeth (Fig. 10 *D*).[21]

Absence of Teeth.—Another condition which has been reported in congenital syphilis is the absence of few or many teeth. This has been questioned.[22] It is important to differentiate between congenitally absent, unerupted, and extracted teeth. In only one of this series of patients were the permanent upper lateral incisors absent. Intraoral roentgenograms did not show their presence in the jaw; there was no history of extraction. This patient, moreover, did not have typical Hutchinson incisors. The absence of teeth (anodontia), partial or complete, is probably on a hereditary basis.[11c]

No particular type of malocclusion occurred with sufficient frequency to be considered characteristic of congenital syphilis, although the open bite was common.[23]

Value of Roentgenograms.—Because clinically the Hutchinson incisor is seen only after the child is 7 years of age, when the incisors erupt into the oral cavity, it has been mistakenly regarded as a symptom of late congenital syphilis. It is only a late manifestation of an early effect. By means of intraoral roentgenograms, the unerupted screwdriver-shaped permanent incisor has been demonstrated as early as the second to the fourth year of life (Fig. 6).[18]

Roentgenograms of the unerupted teeth should be taken routinely with those of bone. Studies of bone are an aid in the early diagnosis of congenital syphilis, but Caffey[24] cautioned that such studies are rarely conclusively diagnostic. Bone, moreover, is subject to resorption and apposition, and the observations are subject to change, especially after treatment. Teeth are not subject to resorption, and the specific findings are permanent. Jeans and Cooke[8a] stated that in general the dental deformities constitute the most important of the stigmas of congenital syphilis on account of their relative frequency. To this statement two other characteristics should be added, namely, the early occurrence and the permanency of the dental dystrophy.

Roentgenographic examination of these teeth is also of value because the true form of the dentinoenamel junction can be determined. Further, because the enamel is wider at the incisal edge than at the cementoenamel junction, the

convergence is more pronounced at the dentinoenamel junction than on the proximal surfaces (Fig. 5). In a recent study of congenital syphilis,[23a] it was found that a greater number of dental stigmas were observed by means of roentgenograms than clinically.

SUMMARY AND CONCLUSIONS

A group of 73 patients with congenital syphilis was studied. Twenty-two (30 per cent) had dental changes characteristic of this disease.

Congenital syphilis, a systemic disease which is exacerbated during the neonatal period and infancy, may affect the particular stage of tooth development active at the time. The effects are different in the gowing deciduous and permanent teeth. The deciduous teeth, active in the formation of enamel, show developmental disturbances in that structure (chronologic enamel aplasia); the permanent teeth, active in morphodifferentiation, show a disturbance of the dentinoenamel junction, with a resulting characteristic dwarfing of the crown.

Consequently, the convergence of the proximal (lateral) surfaces of the permanent upper central incisors (Hutchinson) and molars (Moon, Fournier, Pfluger) should not be confused with disturbances of enamel formation (hypoplasia of the enamel) which occurred at a later time.

However, no correlation was made between the severity of the disease in the neonatal period and earliest infancy and the dental development.

These developmental disturbances of the teeth are permanent and have been found only in congenital syphilis. They can be demonstrated roentgenographically prior to eruption.

Dr. James S. Gold assisted in the clinical part of this study.

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CEMENTOMAS

A Report of 50 Cases

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FIFTY patients with one or more cementomas formed the basis of this study. These constitute most of the cases with cementomas that we encountered at the Columbia University Medical Center over a period of the previous seven years. A few were discarded because of incomplete information.

Our investigation depended on a complete radiographic and clinical oral examination and a history which included the following data: age, sex, race, marital status, present or past systemic diseases or abnormalities including syphilis and gynecologic disturbances. Thirty of our cases were receiving treatment through Vanderbilt Clinic where complete medical histories were obtained.

The diagnosis of cementoma in all instances was based on the following criteria:

1. Radiographic appearance of radiolucency or radiopacity of a periapical area.

2. Clinical observation of the tooth in question as to normalcy in color and negative history of subjective symptoms of pulp abnormality.

3. Pulp vitality tests of these teeth using heat (hot ball burnisher), cold (ethyl chloride) and electrical stimulation (Columbia University pulp tester), to confirm their normal pulp status.

Evidences of periapical abscesses or granulomas, radicular cysts, or other radiolucencies very often simulate cementomas radiographically. These possibilities were eliminated by differential diagnosis based on tooth vitality, history, and clinical symptoms. Periapical cementomas were so designated only when the involved teeth were considered vital. Teeth apparently nonvital, with similar-appearing lesions were discarded.

Cementomas are also known by other designations. Thoma[1] is of the opinion that the term cementoblastoma is more descriptive since this condition arises as a result of a hyperplasia of cementoblasts which causes an osteolysis followed by a deposition of cementum in later stages. McCall and Wald[2] believe that the cementoma is a lesion that has some of the characteristics of localized osteitis fibrosa or fibro-osteoma, and since it occurs at the apex of the tooth it might be called a periapical osteofibrosis.

A cementoma may be defined as a neoplasm of limited growth originating from the cementoblasts of the peridental membrane surrounding the root apex of the tooth. This soft tissue hyperplasia which represents the first stage of cementoma causes osteolysis of periapical bone and is followed in later stages by cementum deposition and hypercalcification.

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Stafne[3] reported thirty-five cases of cementomas of which twenty-five occurred in females. The ages ranged from 23 to 60 years, the average age being about 43 years. Of the seventy-eight teeth involved in this study sixty-nine were mandibular incisors. In a later study, Stafne[4] found that mandibular involvements had a 17 to 1 ratio to maxillary lesions. He found no evidence linking the cementoma with systemic disease and in no instance was syphilis present. Stafne cites Simpson's opinion that incisal trauma may be the exciting causative factor when the necessary individual predisposition is present. However, he concludes that it would be "difficult to explain their presence on the basis of injury." Thoma[1] is of the opinion that the etiology may be constitutional since these tumors are frequently multiple. Dewey[5] believes that both a predisposing constitutional factor and a local exciting factor are necessary for the production of a cementoma.

Statistics (Table I).—The ages ranged from 20 to 68 years. The average age at discovery was about 40 years. Eighty-four per cent were found between the ages of 20 to 49 years. *All patients were females.* Twelve cases were discarded because of incomplete information. Of these, seven were of unknown sex; the remaining five were females but could not be used because other needed information was unobtainable. Thirty-three, or 66 per cent, of the patients were Negroes. The remaining 34 per cent were whites, forming a ratio of almost 2 to 1. Seven patients gave a positive history of syphilis and twenty-one gave a negative history. In the remaining twenty-two patients, complete syphilitic histories were unobtainable. Eleven had one or more gynecologic disturbances; 7 had syphilis; 5 had arthritis; 5 had cardiovascular disturbances; 4 had rheumatic fever; 3 had endocrine gland disturbances; 2 had malignant neoplasms; 2 had gall bladder diseases; 13 had various other systemic ailments as influenza, pneumonia, pertussis, typhoid, etc.

Twenty-eight (56 per cent) had had one or more pregnancies, seven denied ever being pregnant and the remaining fifteen were unavailable for this questioning.

Eighteen patients, or about 36 per cent, presented with a single isolated cementoma, while the remaining 32 patients had 2 or more lesions. Twelve of the 18 isolated lesions occurred about lower anterior teeth, 4 occurred about lower posterior teeth, one occurred about an upper anterior tooth and one about an upper posterior tooth. The largest number of lesions found in a single mouth was 14. The total number of teeth with cementomas was 155.

Forty-three patients (86 per cent) had lower anterior involvements with or without involvements in other regions of the jaw. The remaining 7 patients (14 per cent) had lesions in other parts of the jaws with no lower anterior involvement; 24 patients (48 per cent) had lesions in the lower posterior region. The upper anterior region was involved in ten cases (20 per cent) and the upper posterior region was involved in ten cases (20 per cent). The ratio of mandibular to maxillary cementomas was 7 to 1.

The lesions were classified according to stages in their development by radiographic examination. The classification used is that suggested by Thoma.[1] The number of cementomas in each stage is as follows:

Stage I—The hyperplastic or osteolytic stage was found in 24 cementomas (Figs. 1, 2, 3, and 7).

Stage II—Cementoblastic stage, in which there is active deposition of cementum, was seen in 119 cementomas (Figs. 3, 4, and 5).

Stage III—The radiopaque or the mature, inactive stage with complete calcification was found in 12 cementomas (Figs. 6 and 7).

Cementomas, in more than one stage of development in one patient, were seen in several instances (Figs. 3 and 7).

Analysis of the stages of growth of the cementoma in reference to the age of the patient showed that the youngest patient with a stage I lesion was 25 years old while the oldest was 47 years. In the stage II group the youngest patient was 20 years and the oldest 68 years. In the stage III group the youngest patient was 29 years and the oldest 47 years of age.

Fig. 1. Fig. 2. Fig. 3.

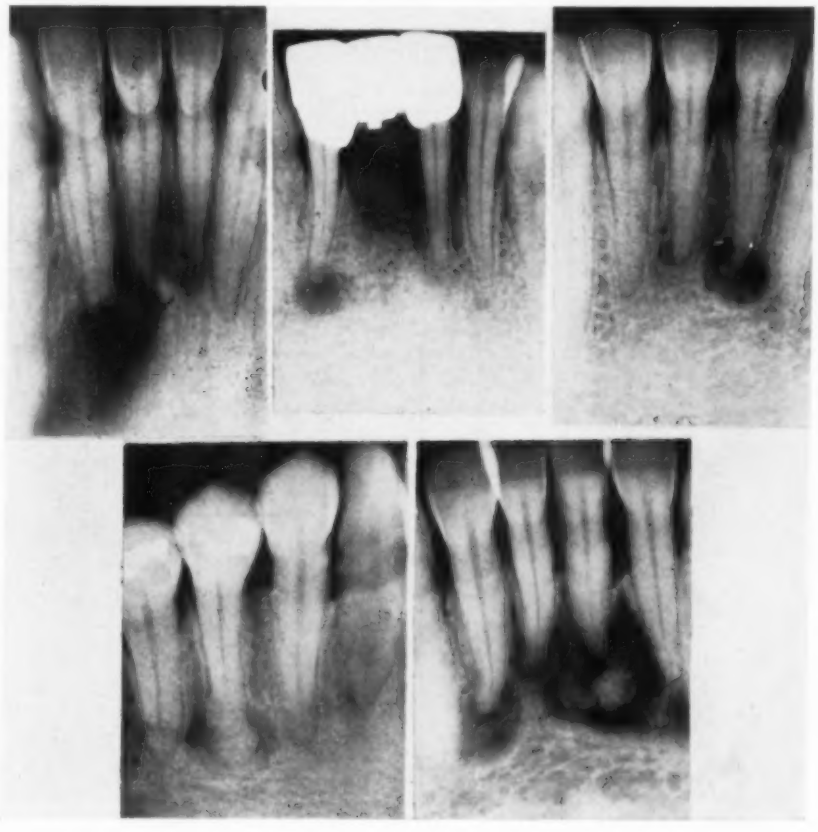

Fig. 4. Fig. 5.

Fig. 1.—Case No. 43 in Table I. Stage I. Earliest observable stage of a cementoma roentgenographically. Lower right central incisor shows thickening of the peridental membrane with beginning proliferation. Lower left central and lateral incisors are more advanced and show a large osteolytic area without any cementum deposition. Note also root end resorption at apices of these teeth which is probably caused by the proliferative tissue. This is not an uncommon finding in connection with cementoma and is an important consideration in differential diagnosis.

Fig. 2.—Case No. 22. Stage I. Shows thickened membrane and osteolytic area at the apical region of lower left lateral incisor. This case is of interest because this tooth is a bridge abutment and might easily be interpreted as a periapical abscess.

Fig. 3.—Case No. 24. Stages I and II. Shows the lower left central incisor in an earlier stage of involvement than the lower right central incisor.

Fig. 4.—Case No. 36. Stage II. Shows an osteolytic area with cementum deposition in the apical region of lower left first premolar.

Fig. 5.—Case No. 10. Stage II. Shows a more widespread involvement.

TABLE I

FINDINGS IN FIFTY CASES OF CEMENTOMAS

CASE NO.	SEX	AGE	RACE*	MARITAL STATUS*	PREGNANCY*	LESIONS				SYSTEMIC
						SINGLE	MULTIPLE	LOCATION*	STAGES IN DEVELOPMENT*	
1	F	41	B	M	?		√	UR 45678; UL 45; LL 345	2	None
2	F	36	B	M	?		√	UL 2; LR 6	2	Headaches
3	F	27	W	M	?		√	LL 23	2	Blurring of vision
4	F	29	B	S	–		√	LR 123; LL 12	2	None
5	F	68	W	M	–		√	UR 12; UL 2; LL 2; LR 6	2	Rheumatic fever; arthritis
6	F	37	B	S	?		√	LR 12; LL 1	2	Arthritis
7	F	49	B	M	+		√	LR 12345; LL 123456	2	Gastric ulcer; arthritis; fibromyoma uterus; hysterectomy; hypoanemia
8	F	37	B	M	?	√		LR 1	2	None
9	F	34	B	M	+		√	LR 12; LL 12	2	Typhoid fever; allergy
10	F	50	B	M	+		√	LR 1234; LL 12	2	Syphilis; typhoid fever
11	F	25	W	S	?	√		LR 2	1	None
12	F	34	B	M	?		√	LR 1; LL 1	1, 2	None
13	F	40	W	M	?		√	LR 1; LL 12	2	None
14	F	33	W	S	?	√		LL 2	2	None
15	F	42	W	M	+	√		LL 4	2	Ovariectomy; fibroid
16	F	33	B	M	+		√	LR 12; LL 12	2	Osteitis fibrosa cystica
17	F	47	B	M	?		√	LL 23	1, 3	None
18	F	36	W	S	–	√		LL 1	2	Arthritis
19	F	35	W	M	+	√		LR 2	2	Cholecystectomy
20	F	48	W	M	+	√		LL 2	2	Cardiac; rheumatic fever
21	F	56	B	M	?		√	UL 6; LR 1237; LL 2367	2	None
22	F	34	W	M	?	√		LR 2	1	None
23	F	37	B	S	?		√	UR 12; UL 36; LR 12367; LL 12378	1, 2	Cardiac murmur; syphilis
24	F	23	B	M	+		√	LR 1; LL 1	2	Eczema
25	F	43	B	M	–	√		LR 1	2	Syphilis; fibroid; hysterectomy
26	F	57	W	M	+		√	LR 123; LL 124	3	Diabetes mellitus; hypertension

**Race:*
B = black
W = white

Marital Status:
M = married
S = single

Pregnancy:
? = information unobtainable
– = none
+ = one or more

Location of Lesions:
UR = upper right
UL = upper left
LR = lower right
LL = lower left

Stages in Development:
1 = osteolytic
2 = cementoblastic
3 = mature

TABLE I—CONT'D

CASE NO.	SEX	AGE	RACE*	MARITAL STATUS*	PREGNANCY*	LESIONS				SYSTEMIC
						SINGLE	MULTIPLE	LOCATION*	STAGES IN DEVELOPMENT*	
27	F	34	B	M	+		√	LR 1; LL 1	1	Syphilis; carcinoma of cervix
28	F	36	B	M	+	√		LR 1	2	Fibroid; maxillary sinusitis; salpingitis; hysterectomy; typhoid fever
29	F	35	W	M	+		√	UL 1; LR 12; LL 12	1, 2	Hypertension
30	F	52	W	M	+		√	LR 1; LL 12	2	Myelogenous leucemia
31	F	40	B	M	+	√		LL 2	2	Syphilis; cholecystitis
32	F	35	B	M	+		√	LR 367; LL 246	2, 3	Cyst of Bartholin's glands
33	F	25	B	M	+		√	LR 123; LL 123	2	Fibroid; hyperthyroid
34	F	35	W	M	+		√	LR 1; LL 1	3	None
35	F	30	B	M	+		√	UR 2; UL 2; LR 123; LL 12	1, 2	Influenza; rheumatic fever
36	F	43	B	M	?	√		LR 4	2	Atrophic rhinitis; syphilis
37	F	23	B	M	+		√	LR 3; LL 1	2	Puerperal sepsis
38	F	50	B	M	+		√	LL 12	2	Carcinoma of breast; osteoarthritis; hypertension; ovariectomy
39	F	39	B	M	+		√	LR 12; LL 12	1	Fibroid; hysterectomy
40	F	29	W	M	?	√		LR 7	3	None
41	F	51	B	M	+	√		LL 1	2	Several abortions
42	F	27	B	M	+		√	LR 12	2	None
43	F	30	B	M	−		√	LR 12; LL 3	1	Arthritis
44	F	31	B	M	−		√	LR 12	2	None
45	F	34	B	M	+		√	LR 1; LL 1	2	Pertussis; pneumonia
46	F	35	B	M	+	√		LR 1	1	Syphilis
47	F	31	B	M	+		√	LL 23	2	Fibroid; puerperal mastitis
48	F	32	B	M	+	√		UL 5	3	None
49	F	20	W	S	−	√		LR 7	2	None
50	F	37	W	M	+	√		UR 2	3	Rheumatic fever; menstrual dysfunction

In a number of cases we were able to follow radiographically the developmental cycles of cementomas from the osteolytic to the mature, radiopaque stage. It was found that these lesions are slow-growing. In one instance thirteen years were required for full development of a cementoma from stage I to stage III (Figs. 8 through 13). In many instances little increase in calcification was noted after three or five years following discovery of the lesion.

None of the fifty patients gave histories of trauma to the involved teeth.

Of the 155 teeth in this series only 4 were found to have carious cavities and 12 had fillings or crowns.

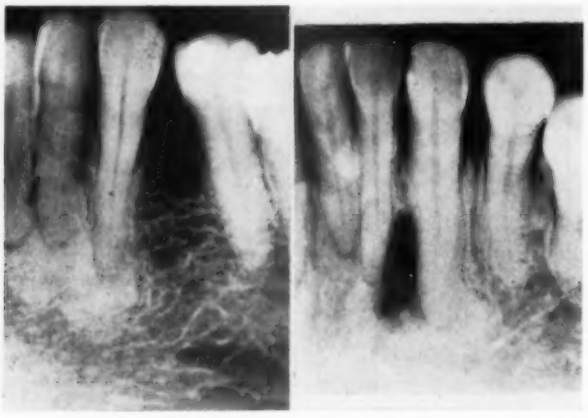

Fig. 6. Fig. 7.

Fig. 6.—Case No. 26. Stage III. Shows the former osteolytic areas completely filled in with cementum.

Fig. 7.—Case No. 17. Stage I and Stage III. The lower right lateral incisor shows an osteolytic area and the lower right cuspid shows a radiopaque area in the apical region.

Fig. 8. Fig. 9. Fig. 10.

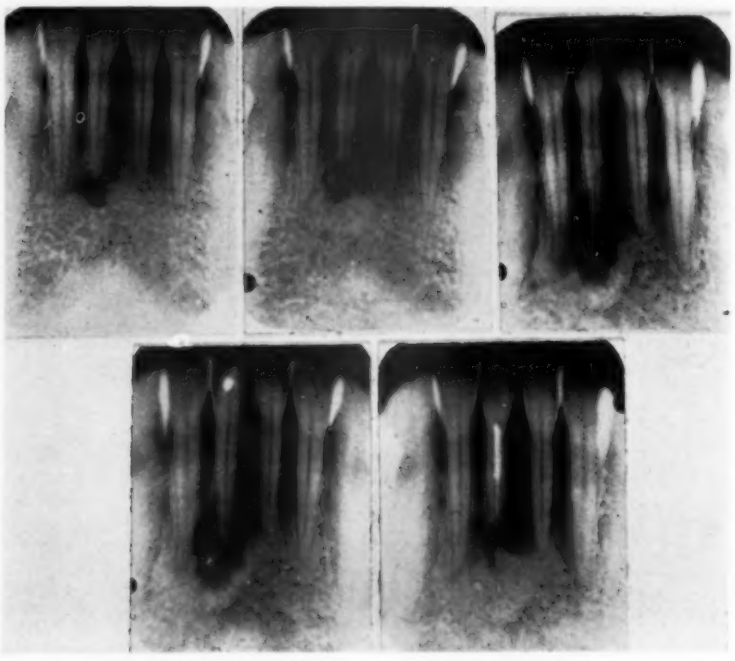

Fig. 11. Fig. 12.

Figs. 8 through 13 are of Mrs. R. H., aged 22 years, white, who presented with a radiolucent area about the lower left central incisor, on May 19, 1928 (Fig. 8). Condition was undiagnosed at this time. However, it was kept under observation and on April 4, 1931, a second x-ray was taken (Fig. 9) which showed an early Stage II cementoma. Fig. 10 is from an x-ray taken Aug. 19, 1936, and shows a more advanced Stage II. Fig. 11 is from an x-ray taken Oct. 12, 1936, at which time an attempt was made to test the tooth for vitality, by drilling into it. An accidental pulp exposure was made. The pulp was capped and retained. About one year later, a devitalization and root amputation was done (Fig. 12) and the excised cementoma was examined histologically. Clinical diagnosis of cementoma was confirmed.

DISCUSSION

The importance of the 2 to 1 ratio of Negroes to whites found in our group of patients assumes much greater significance when we study the attendance records at the Dental Clinic. During the period from Jan. 1, 1938, to July 1, 1942, the average attendance at the clinic was 82 per cent white and 18 per cent Negro, which is approximately a 4 to 1 ratio of whites to Negroes, making a real difference in the frequency of occurrence of cementomas between black and white races as 8 to 1.

Because of the small number of positive syphilitic histories in our group, the possibility that syphilis plays a role in the etiology of cementoma is remote. There is no direct correlation between the incidence of cementoma and the present or past history of syphilis. Nor is there any correlation between the occurrence of cementomas and any other systemic disease.

The fact that the cementoma occurred as a multiple lesion so often in our group makes the theory of local trauma untenable especially so since we were unable to discover any alleged traumatic agents. Similarly, the fact that only 12 teeth of 155 had cavities or restorations, negates their possible etiologic role. A systemic background is a more likely explanation for these tumors.

Fig. 13.—Taken on Jan. 27, 1941, thirteen years after the first x-ray, shows complete regeneration. (This case is presented with the permission and through the courtesy of Dr. Harry Seldin.)

The significance of the mandibular-maxillary ratio is undetermined at the present time. Future investigation of the underlying causes of cementoma may elucidate the reasons for the tendency of cementomas to occur in the anterior portion of the mandible.

The most interesting finding in our study was the factor of sex. Since all these growths were found in females, the female hormones assume an important role in etiology. The ability of the estrogens to stimulate connective tissue has been reported by one of us.[6, 7, 8] The effects of the estrogens on mesenchymal structures have been extensively reported in the literature.[9, 10, 11, 12, 13, 14, 15] Just what the relationship here may be is, of course, unknown.

Another hormonal consideration in etiology is the character of the lesion in its incipiency. The lesions begin as a fibrous tissue growth with cementum deposition later in the course. This has been observed repeatedly in histologic sections of these growths. In a previous study we reported the ability of the

gonadotropic hormone of pregnancy urine (Prolan) to stimulate the formation of the so-called "pregnancy tumors" which are also fibrous connective tissue hyperplasias.[16, 17]

The thought, then, presents itself that, since 84 per cent of these women were married and only 14 per cent denied pregnancies, this hormone may be involved in the etiology of these growths at the tooth apex as well as at the gums. Moreover, the fact that all of our observations occurred in women, makes plausible the hypothesis that the hormones involved in the female sex are in some way etiologic agents.

SUMMARY AND CONCLUSIONS

1. The cementoma is a definite clinical entity which can be distinguished from other lesions by certain tests outlined.

2. *It occurred exclusively in women in our group.*

3. It is much more frequent in Negroes than in whites, (about 8 to 1).

4. Caries or restorations in teeth and trauma bear no demonstrable relationship to the incidence of cementomas.

5. No systemic diseases were found which could be correlated with cementomas. Our findings seem to indicate that an endocrine and/or metabolic or racial explanation of etiology may ultimately prevail.

We wish to express our appreciation to Mr. Francis Bodkin, for his efforts in gathering some of the material contained in this report.

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Note of Interest

Major Thomas J. Cook of the Editorial Staff of the Oral Surgery Section of the Journal was promoted to the rank of Lieutenant Colonel on Jan. 14, 1943, and is now serving with the 20th General Hospital in India. The address is Lt. Col. Thomas J. Cook, 20th General Hospital, A.P.O. 689, c/o Postmaster, New York, N. Y.

Editorial

Teaching Stomatology (Dentistry) in Medical Schools

In 1925, when this writer founded the *First Post-Graduate School of Stomatology* chartered by the University of the State of New York, one of the stated purposes was:

> . . . to give to graduated and duly licensed physicians and dentists an opportunity to obtain systematic instruction in stomatology in the United States, the same instruction that is afforded medical practitioners and stomatologists in the European Stomatological Schools. . . . The school aims to draw to its classes those practitioners in medicine and its specialties and dental practitioners who wish to keep abreast of the progress of stomatology in relation to general medicine.[1]

The main aim of that institution was to bring about effective medicodental cooperation through one of the most useful social instruments, namely, *education*.[2] Although no appreciable advance has been made in this direction during the past two decades, some steps ahead have been taken from time to time, in a somewhat tortuous fashion.[3] This writer has not been a passive bystander in this area of medicodental endeavor but has made a conscious effort to contribute his share to its progressive development.[4] Let us glance at some of these events with a view to noting vital problems that now confront the dental profession with respect to teaching stomatology (dentistry) in the medical schools of America.

The purpose of this discussion is twofold: First, to take note of a current trend in medical education in favor of including *formal* instruction in stomatology (dentistry) as part of the undergraduate curriculum. Second, to point out the need for giving the proper direction to this trend so that it may not deviate from its intended course and so that it may attain the desired results. Although our discussion deals with the problem of cooperation between dental and medical practitioners, our attention will at this time be focused on the *educational* phase.[5]

Let us first inquire about the following: Would instruction of medical students in the principles of dental science be beneficial: (a) to them as practitioners of medicine and the specialties of medicine, (b) to their effective cooperation with dentists and dental specialists, (c) to raising the quality of health service in general? The oral internist and oral surgeon would without hesitation say "yes." Dentists, viewing the situation in more practical terms, would see no objection to cooperating with *dentally* educated physicians. Before the present war, and in these days especially, a considerable number of enlightened physicians and medical specialists would have shared this opinion. The medical scientist and the medical educator, who look at the health picture

in broad scientific terms and see the social implications and public significance of this step, would certainly give their approval. Theoretically there is no problem, but in practice dental and medical educators have always faced this age-old problem. They sought a solution. The findings of our latest study (1943) indicate that we are on the path to solving it.[6]

We should bear in mind that *formal* instruction in stomatology has in store for us newer problems which should now be carefully weighed, for we are at the parting of the ways in this area. Conditions today are not what they were in 1923 when dentistry and medicine were not so far advanced, and social conditions were not as complex as they are today. Let us briefly review what has transpired since then.

In 1933, when a course in preventive medicine was introduced at the New York University Medical School, this writer called attention to that event:

> A most notable and exemplary step has been taken by New York University toward bringing medicine and dentistry together through the means of teaching medical students their newer social responsibilities in the practice of prevention. It was through the efforts of Dr. John Wyckoff, Dean of the Medical School, and Dr. Allen T. Newman, Dean of the Dental School, that five lectures on the fundamentals of dentistry were included in the course of preventive medicine given by Dr. William H. Park. . . . The oral topics covered in this year's course include The Relationship Between Oral and Systemic Disorders given by Dean Newman; Oral Surgery by Professor Leo Winter; Dental Caries, Pyorrhea, and Focal Infection by Dr. Alfred J. Asgis; Orthodontia by Professor Lester Stanton, and Interpretation of Dental Radiograms by Professor Greenfield. The importance of teaching to future medical practitioners the fundamentals of stomatology in the course of preventive medicine will be more readily appreciated.[7]

In 1924-25, only 9 medical schools offered instruction in stomatologic subjects. In 1929-30, 15 out of 81 medical schools in the United States gave instruction in oral hygiene, clinical dentistry, oral surgery, or the fundamentals of stomatology in the regular curriculum. The total time allowance to dentistry in the four-year medical course averaged eight clock hours. In 1940, of the 86 medical schools in the United States and Canada surveyed, 46 (53 per cent) replied, and of this number only 20 devoted any time to instruction in dental subjects; the hours of instruction in this group ranged from 3 to 27 per year.[8] According to the findings of this year's survey (1943), of the 69 schools that replied (out of a total of 86), 31 offer instruction in dentistry, stomatology, and related subjects. Of the 31 schools, 14 offer formal courses, and 17 give informal instruction.

Since 1940 two schools have introduced courses under the heading of "stomatology" while others give instruction in oral surgery, dental pathology, dental hygiene, and related subjects in connection with the various medical and surgical departments. The informal dental courses are offered by various department heads of the medical school, sometimes by dental faculty members. There is apparent a trend in medical education toward dental instruction—formal and informal.

Those of us who are engaged in the scientific and educational advancement of oral medicine and oral surgery are faced with some educational and professional problems that call for immediate consideration. Should medical schools, with all due respect for the expert counsel they have been and may be receiving, be left to individual guidance and do "the best they can"? Or should dental instruction in medical schools be aided by group thinking and the cooperative and studied guidance of those active in and vitally concerned with this specialty of health service?

Before we go any further, it would seem proper to indicate the meaning in which the term "stomatology" (now that the ban on its use appears to have been lifted) will be used here, both in relation to medical students and dental practitioners. For the benefit of physicians and dentists who were not clear on the meaning of stomatology,[9] the writer stated in *Clinical Medicine & Surgery:*

> The *science* of stomatology is the study of the normal and abnormal, in structure and function, of the mouth organs, either as cause or effect of local or constitutional disturbances or both within the mouth, the body or other organs. The *art* of stomatology refers to the means, methods and procedures employed by the mouth physician and surgeon to prevent oral disease, to maintain mouth health and to restore to normal function an impaired organ. The practitioner who engages in the practice of stomatology as a whole or in some of its phases is called a *stomatologist*.[10]

This is not to infer that the medical student who has been taught the principles of stomatology in one of his undergraduate courses is qualified for the practice of dentistry, any more than he is qualified in ophthalmology, rhinology, gastroenterology, etc., after attending some lectures or clinics dealing with these subjects. Only the dentist who has been educated and trained for the profession of dentistry, having received a basic education in the medical sciences and in this specialty, is qualified to engage in the practice of stomatology (dentistry). We who are working in the medical and dental professions are aware of the need for repeated emphases on this point. Confusion about the status of dentistry has arisen in the past two decades as a result of the many plans proposed for reform in dental education.[12]

The writer is of the opinion that only *two* systems of dental education have so far been evolved and are functioning in modified forms today, namely, the *stomatologic* and the *autonomous*.[13] The supporters of the level-technician scheme delude either themselves or others, perhaps unknowingly, when they believe that they are proposing a new and third system of dental education. By removing from their schemes the *stomatologic* plan, their projects turn out to be an ill-conceived approach to solving the problem of training and licensing dental technicians. Such a roundabout approach to a serious problem will undoubtedly prove detrimental to the public and the health professions.[14, 15]

Let us be more specific. Educationally speaking, would it not be more fruitful, in the interest of both medicine and dentistry, to endeavor to formulate some concrete approach to the problems of course content, the determination of areas of the dental field that should be included in medical instruction, the

setting up of criteria for the selection of subject matter, the choice of methods of teaching, the instructional aids, methods of evaluating outcomes of instruction, etc.? These are but a few of the curricular and instructional problems.

No less important are the administrative aspects of the issues before us. What should be the minimum number of hours for the course? Should it be didactic and (or) clinical? Who should teach it, a dentist or a physician? Who should determine the teaching load? What should be the qualifications of the dental teacher in medicine?

Mention should be made of the fact that in Italy and in the Soviet Union medical students must pass an examination in the principles of stomatology as a requirement for graduation. The aim of this instruction in these countries is clearly defined by concept—accepted by the health professions, the public tradition, and the law. Considering that under our autonomous system of dental education many dentists have been led to believe that dentistry is largely mechanical, and the fact that the public has not been convinced to the contrary, conditions here and abroad are hardly comparable. Dental considerations before the Pepper Committee and the projected American form of the Beveridge plan (and other postwar proposals) bear watching in the light of current political trends.[16] In some European countries unqualified persons were admitted to dental practice under radical social changes.[17] Progressive dentists and physicians will do well to be on the alert inasmuch as progress may be impeded by forces emerging from at least three channels: (1) by commercial opportunists who stop at nothing to exploit for financial gain a promising idea, and care little about ideals; (2) by autonomists in dentistry who are suspicious of any change; and (3) by the more sinister of these groups who support a "level-technician" scheme which may be offered to the public under a variety of subtle patterns.[18, 19]

To prevent such unwholesome occurrences, and as a partial answer to the educational questions raised, the following suggestions are offered with respect to course content and teaching method.

1. *Course content.* Subject matter should be selected from the fundamental oral sciences, oral medicine, oral surgery and *oral restorations* to enable the medical student to get a general understanding of the *theory* on which rests present-day dental practice. Restorative dental procedures can and should be interpreted in terms of these *health* objectives. No matter what method of teaching is employed, the teacher should see to it that no trace of the mechanical concept of dentistry enters the medical thinking of the undergraduate student. *All* of dental science, including prosthesis, should be interpreted in biomedical terms and correlated with other fields of medical knowledge.[20] As far back as 1936 in his introductory lecture on "Dental and Oral Disorders in Relation to Systemic Disease, With Emphasis on Dental Caries, Pyorrhea, Focal Infection and Orthodontia," which was given before senior medical students of New York University, the writer used *Clinical Diagnosis of Oral Diseases* (1935) by Hayes. Today, with the availability of such representative works as that on Oral Pathology (1942) by Thoma, and similar contributions by others, the problem of content is much less complicated.

2. *Case-discussion method of teaching.* This method would seem to be most suitable in instructing medical students in stomatology. Here, the oral case represents the unit of instruction. It provides the medical student, and the dental teacher of stomatology, with opportunities for the discussion of principles and their application to diagnosis, treatment, and follow-up. The student is immediately set into a sympathetic attitude toward the oral patient, and toward the whole of dentistry (stomatology). He integrates conceptually into a unified whole oral problems with other special problems of health service. He thus approaches their solution in the light of their respective *values.* In this educational setup, clinical dentistry and stomatologic science may anticipate receiving considerate intellectual treatment by the medical student of today who is the practitioner of tomorrow.[21]

But this is not the whole story. Even the best of programs is likely to fail in its objective unless there is a philosophy behind the effort. In this respect the administrative phases must be handled by the deans of dental schools and deans of medical schools who possess farsightedness and social vision. Let no one group in the health professions be tempted to secure for itself benefits at the expense of others. During the impending period of postwar reconstruction we must beware of those who will suddenly emerge and rush in with unwholesome proposals under the guise of socioeconomic reform intended to help dental cripples in the low-income groups. The gradual and steady awareness of health service practitioners regarding the need for steady public betterment should deter us from supporting misleading projects.[22] Let us not repeat the mistakes that were made by shortsighted dental and medical leadership in Austria, Germany, and other European countries prior to the rise of the Nazi regime. More enlightenment and less withholding of information are needed. Let us stop confusing the issues of autonomous and stomatologic education with plans for the *dismemberment* of dentistry.[23]

Medical students should be taught the principles of stomatology (dentistry) in order to qualify them as collaborators with dentists in rendering an improved health service to the public. To that end, we have no doubt that public-spirited dentists and physicians will give their hearty support.[24]

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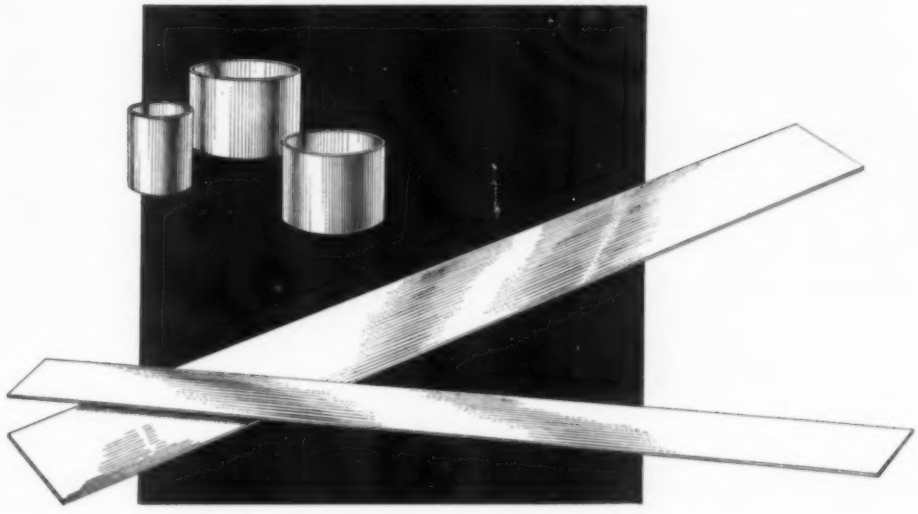

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